VOLUME 66 • JULY 1957 • NUMBER 1

A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

Special Study in the Use of Cold Vaccine

J. Charles Dickson, J. P. Abbott, and Robert Davis

The Ear in Severe Malformations of the Head

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Direct Adenoidectomy

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The Dry Physiologically Perfected Fenestra Nov-Ovalis Technique

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A Study of One Hundred Cases of Neck Dissection

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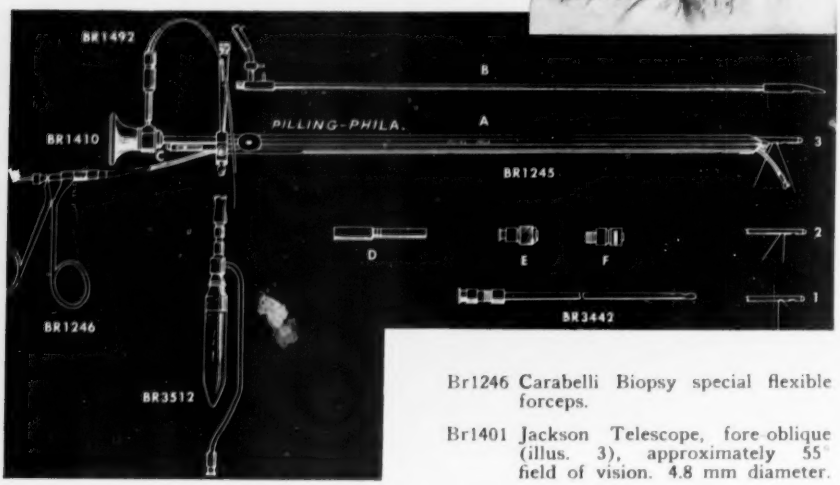

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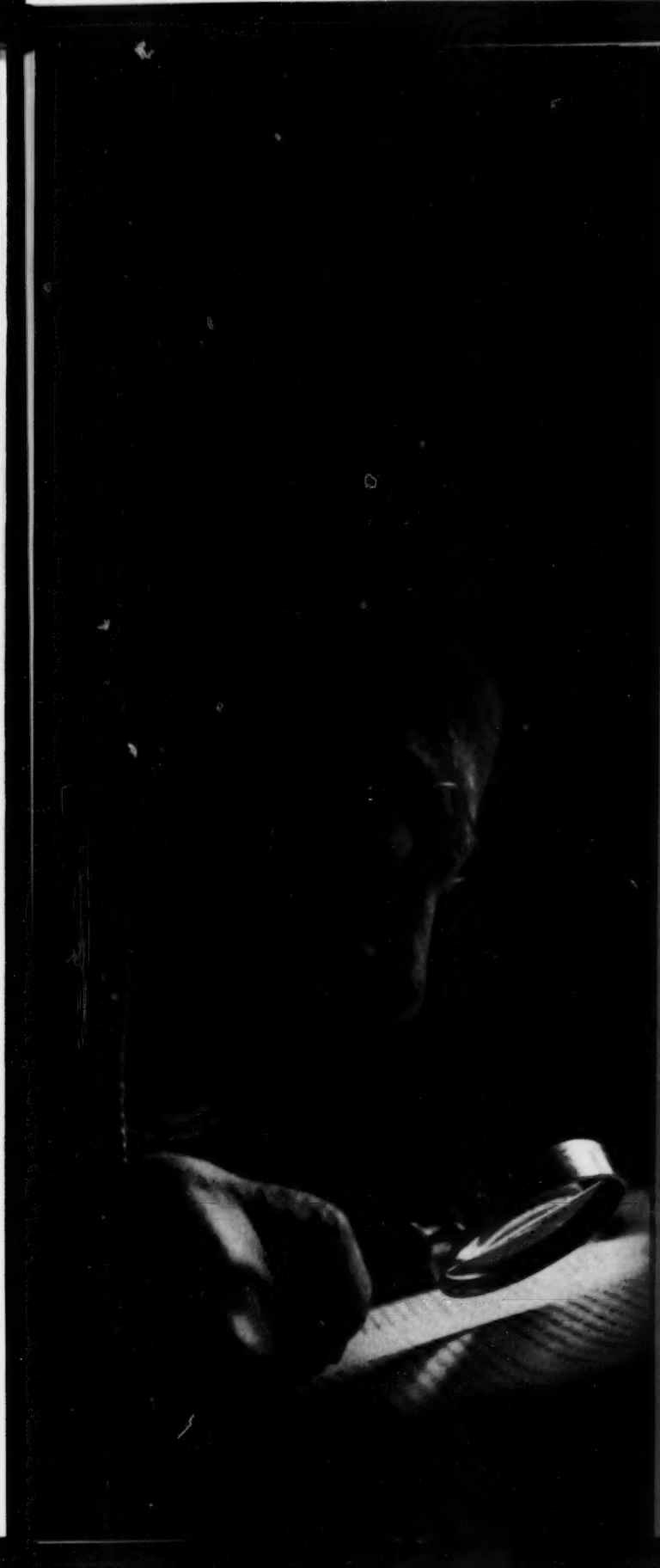

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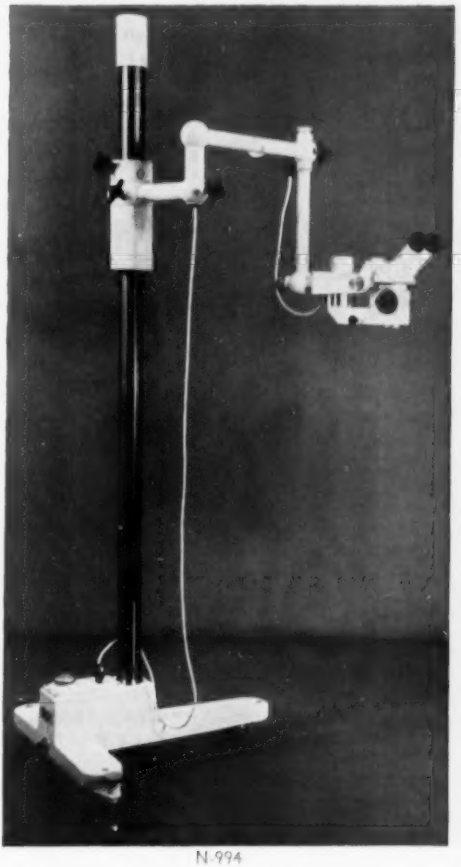

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OTOLARYNGOLOGY

Special Study in the Use of Cold Vaccine

J. CHARLES DICKSON, M.D.; J. P. ABBOTT, M.D., and ROBERT DAVIS, M.D., Houston, Texas

Introduction

Since the introduction of antibiotics to the medical armamentarium little attention has been given to the value of bacterial vaccines for the prevention and treatment of the common cold. The use of antibiotics has greatly reduced the serious complications of upper respiratory infections. We see very few serious complications such as mastoiditis, meningitis, extensive sinus infections, etc. However, it is our feeling that there has been a definite increase in the frequency of upper respiratory infections. A patient develops an acute cold, with secondary bacterial invaders producing the disagreeable symptoms that go with an acute cold. He or she is given intensive antibiotic or sulfonamide therapy. The patient recovers from this upper respiratory infection only to develop another within one to three weeks. This is repeated over and over again until the patient seems to have a continuous upper respiratory problem. It is our feeling that the use of antibiotics and sulfonamide prevents the patient from developing active immunity. If some means could be found to stimulate immunity or antibody production at the same time the patient is being relieved of his disagreeable cold or serious complications by the antibiotics and sulfonamides we would be serving our patients much better than we are at the present time.

Accepted for publication Jan. 3, 1957.

From the Baylor University College of Medicine and the Methodist Hospital.

This study was started with the above fact in mind. The study was to evaluate the ability of a locally prepared pooled bacterial vaccine to stimulate the production of nonspecific antibodies, thereby preventing the development of colds and bacterial sequelae. Careful controls and selection of patients were maintained as well as possible throughout the study. During this study a chance to evaluate any change in the protein-production pattern of the patients was afforded. This latter portion of the investigation was done through the facilities of the paper electrophoresis technique. A report will be made later on this phase of the investigation.

Materials and Methods

The pool vaccine used in this experimental survey was composed of organisms which were isolated from patients complaining of acute and chronic nasopharyngitis, tonsillitis, sinusitis, and otitis media. These organisms were isolated in the usual manner by using blood agar, brain-heart infusion broth, and fluid thioglycollate medium, and in the latter part of the isolation a special staphylococcal medium was used. After isolation these organisms were grown in pure culture, harvested, and suspended in saline. Before this vaccine was used in this study the following numbers of various strains of organisms were included in the pool stock vaccine from which these injections were given: 41 strains of Staphylococcus albus, 29 strains of Staphylococcus aureus, 7 strains of Micrococcus tetragenus, 31 strains of Streptococcus pyogenes, 63 strains of Streptococcus viridans, 9 strains of Diplococcus pneumoniae, 50 strains of Neisseria pharyngitis, 5 strains of Proteus vulgaris, 8 strains of Klebsiella pneumoniae, 17 strains

of Corynebacterium hofmannii, 7 strains of the Hemophilus species, 49 strains of gamma streptococci, and 2 strains of Pseudomonas aeruginosa.

After preparation of this vaccine the organisms were washed repeatedly in sterile saline, were killed with 1:10,000 thimerosal (Merthiolate), and standardized grossly by the nephelometer method; and the final standardization was based upon Kjeldahl's method of total protein nitrogen. The vaccines were checked for sterility by anaerobic and aerobic techniques for 96 hours' incubation and for toxicity by mouse inoculation of 1 cc. interperitoneally of the 0.195 mg. nitrogen concentration. A concentrated pool vaccine of these organisms in equal portions was then prepared, and this concentrate was stored in the Deepfreeze at —80 degrees. From this stock concentrate all vaccines for this study were prepared.

At the beginning of this study one of us prepared a code which consisted of white, yellow, and blue cards with the information printed on them shown in the Figure. Each patient receiving this injection was given a code number and a card in rotation. There was no knowledge by the ones administering this vaccine or the patients as to what color card designated what concentration of vaccine. The white cards were used for control studies, and this bottle contained only isotonic saline. The yellow cards were used for those receiving the very dilute vaccine, which contained 0.00002 mg. of nitrogen per milliliter. The blue cards represented a vaccine which contained 0.0002 mg. of nitrogen per milliliter.

Two different concentrations of each of the vaccines and control were than given to the clinical staff for injection. The second bottle of each vaccine contained 100 times the nitrogen concentration of Vial 1, thus giving the yellow bottle a concentration of 0.0002 mg. of protein per milliliter and the blue bottle 0.02 mg. of nitrogen per milliliter.

Selection of Patients and Methods of Injections

This vaccine was used on members of the staff of four institutions, the Methodist Hospital, St. Luke's Hospital, Texas Children's Hospital, and Baylor University College of Medicine. Volunteers were solicited from the employees and students of the four institutions. All those volunteering were given code numbers and one of the three colored cards in rotation. The card color designated which bottle of vaccine or control the persons would be receiving. The injections were given subcutaneously by the nursing staff under supervision of the investigators, starting in October and finishing in March.

At the beginning of the study each person who volunteered for the injections was carefully questioned as to numbers of colds he had the previous year and his known allergies, sensitivities, and chronic upper respiratory diseases. This material was recorded on his card as part of the permanent record. At the time of the first injection these patients each had 10 cc. of whole blood drawn for determination of total protein and protein-separation patterns.

There were 425 patients when this vaccine survey was begun. Of the beginning number, 246 were considered to have had a sufficient number of injections to be of value in this survey. It was felt by the investigators that a person should have had a minimum of five injections to be evaluated in this study.

NAME:
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JOB CLASSIFICATION: WORK HOURS:

Vial #	Date Given	Dose Sched.	Dose Given	COMMENTS AND REACTION
Y1		.1 cc.		Skin test
Y1		.1 cc.		
Y1		.2 cc.		
Y1		.3 cc.		
Y1		.4 cc.		
Y1		.5 cc.		
Y1		.6 cc.		
Y1		.7 cc.		
Y1		.8 cc.		
Y1		.9 cc.		
Y1		1.0cc.		
Y2		.1 cc.		
Y2		.2 cc.		
Y2		.3 cc.		
Y2		.4 cc.		
Y2		.5 cc.		

Injections were carried out along the following schedule, with certain exceptions: For the first injection, 0.1 cc. of the vaccine being used was injected subcutaneously. Following that, at five- to seven-day intervals, an increasing dose of 0.1 cc. was administered until the patient developed evidence of generalized or localized reaction. If either of these developed, the vaccine was left at the previous week's dosage or was dropped back 0.1 or 0.2 cc.

At each visit by the volunteers, they were carefully questioned as to whether the last injection had produced any localized or generalized symptoms, with particular reference to generalized muscle ache and pain, joint pain, skin tenderness, chills, chilly sensation, headaches, etc., and as to whether the arm became excessively sore following the injection. If the patient had developed an upper respiratory infection since the last injection, the next injection was not given but a record was made on his card of this development. Following recovery from the upper respiratory infection the patient began his vaccine dosage at the same level at which it was discontinued. When a volume of 1 cc. had been reached in each of these patients, the second concentration was then begun with 0.1 cc. and progressing upward by 0.1 cc. each week.

At the end of the study all patients completing the vaccine survey were questioned as to the number of colds they had developed during the period of the trial; how the vaccine affected them, and if there were any untoward or unwanted side-reactions.

At the end of this survey it was felt that there was a sufficient number of patients who had completed the course to give some evidence as to the helpfulness of this vaccine in preventing or modifying upper respiratory infections and the common cold.

Protein Electrophoresis

Ten cubic centimeters of blood was drawn from each patient prior to his first vaccine injection. This was allowed to clot, separated as quickly as possible, and the serum quickly frozen.

At a later time the serum was thawed and paper electrophoresis carried out. Protein separation was carried out on the Spinco Model R paper electrophoresis system. In the separation 0.008 ml. of serum was used, and this was run through a buffer which contained diethylbarbituric acid and sodium diethylbarbiturate. This buffer has a pH of 8.6, and the system was run at 5 ma. for 16 hours. Following drying in the oven the strips were stained by the bromophenol blue method and run through the analytrol, after which calculations for per cent and total amounts of albumin, and alpha$_1$, alpha$_2$, beta, and gamma globulins were made. The total proteins on each of these sera were run by the biuret method, and the percentages of the other fractions are based upon this absolute value of total protein.

At the termination of the series of vaccine injections, each patient had blood drawn, which was again submitted for protein electrophoresis. The results of these before-and-after studies are to be tabulated and reported at a later date.

Results

The results of this study have been carefully tabulated in Table 1. Of the total number of patients beginning these injections, only 246 completed a sufficient number of injections to qualify for evaluation in this study. There were 84 patients receiving the dilute vaccine, 76 patients receiving the more concentrated vaccine, and there were 86 patients which received the saline injections. There were 13.2 injections per case with the dilute vaccine, 13.1 injections per case of the concentrated vaccine, and 12.1 injections per case of the saline control. There was no reaction in 60.7% of the patients who received dilute vaccine, in 71% of the patients who received concentrated vaccine, and in 60.3% of the patients who received the saline control. There were no upper respiratory infections

TABLE 1.—*Data on 246 Patients Who Received Injections*

	Type of Vaccine		
	Yellow 0.002	Blue 0.02	White Saline
No. of cases	84	76	86
Av. no. inject. per case	13.2	13.1	12.1
Cases with no reaction			
Number	51	54	60
Per cent	60.7	71	69.8
Cases with no URI			
Number	33	31	36
Per cent	39.3	40.8	41.9
No. colds in all cases			
Total No.	83	97	107
Colds per case	1.6	2.2	2.1
Cases with reactions			
Number	33	22	26
Per cent	39.3	29	30.7
Cases with URI			
Number	51	45	50
Per cent	60.7	59.2	58.1
Results			
Better			
Number	40	35	38
Per cent	47.6	46.1	44.2
Worse			
Number	9	9	13
Per cent	10.7	11.8	15.1
Same			
Number	20	19	23
Per cent	23.8	25	26.7
No information			
Number	15	13	12
Per cent	17.9	17.1	14

in 39.3% of the patients who received dilute vaccine, in 40.8% of those who received the concentrated vaccine, and in 41.9% of those who received the saline control. There were 1.6 colds per case of the patients who received the dilute vaccine, 2.2 colds per case of those who received the concentrated vaccine, and 2.1 colds per case of those who received the saline control. There was some form of local or general reaction in 39.9% of the dilute-vaccine patients, in 29% of the concentrated-vaccine patients, and in 30.7% of the saline (control) patients. Of those who developed upper respiratory infections, there were 60.7% in the group which received the dilute vaccine, 59.2% in the group which received the concentrated vaccine, and 58.1% in the group which received the saline control.

Of the patients receiving the dilute vaccine, 47.6% commented that they were better than in previous years, 10.7% stated they were worse than in previous years, and 23.8% stated they could see no change in the status of their upper respiratory infections due to the vaccine. Of the patients receiving the concentrated vaccine, 46.1% stated that they were better than in previous years, 11.8% stated they were worse than in previous years, and 25% said there was no change. Of the saline (control) patients, 44.2% stated that they were better than in previous years, 15.1% stated they were worse than in previous years, and 26.7% stated there was no change.

Comment

The above results show that this vaccine either in dilute or standard strength did not give any more protection than the saline placebo.

An exhaustive review of the literature was covered for the last 16 years concerning the use of bacterial cold vaccine. This review was very revealing and interesting. In the review all agreed that the virus was the primary cause of upper respiratory infections but that the disagreeable symptoms and complications were caused by bacteria (Dwyer,[12] Andrewes,[2] Cecil,[6] Walsh[32]). In reviewing the articles it was found that the various authors almost split 50-50 in their opinions on the value of the vaccine. The following authors felt that it was of value: Goldstein,[14] Kinghorn,[18] Kolmer,[20,21] Solis-Cohen,[9] Baird,[3] Wilkins,[33] Cheney,[8] Sangster,[29] Malone,[24] and Adams.[1] The following investigators felt the vaccine was of little or no value: Diehl,[10,11] Ojala and Kortekangas,[25] Wilson,[34] McGee,[23] Hunnicutt,[16] Smith,[28] Baetjer,[5] Rammelkamp,[27] Layton,[22] Veasey,[31] Hussey,[17] Gray,[15] Bethea,[4] and Proetz.[26] A status report[30] also stated that the vaccine was of little value.

In another series, of 69 selected patients who were given a stock autogenous vaccine prepared very similarly to the above, better results were obtained. Table 2 summarizes the findings in this series of cases. The vaccine was prepared for the following reasons: colds (33 cases), sore throats (12 cases), ear infections (19 cases), and other infections (18 cases). The vaccine was given for an average of 8.9 months. Nine, or 13%, had no improvement. Seventeen, or 24.6%, had slight improvement. There was moderate improvement in 25, or 36.3%. There was marked improvement in 18, or 26.1%.

The results from the mass use of the cold vaccine and the results from the selected use of the vaccine thus differed considerably. It is our feeling that in selected

TABLE 2.—*Results of Use of Stock Autogenous Vaccine in Sixty-Nine Selected Cases*

No. of cases	69
Reasons prepared	
Colds	33
Sore throats	12
Ear infections	19
Other infections	18
Average length of time given (months)	8.9
No improvement	
Number	9
Per cent	13
Results	
Slight improvement	
Number	17
Per cent	24.6
Moderate improvement	
Number	25
Per cent	36.3
Marked improvement	
Number	18
Per cent	26.1

cases where more careful supervision is possible the results are much better. This may also explain the wide divergence of opinion on the value of the vaccine among the various authors.

Another fact noted was that in some people the vaccine could not be given in even a dilute dose. It was necessary to reduce the dosage to 1:10,000 dilution to keep from getting reactions. These were the cases who on receiving the standard or dilute dose in the mass inoculation stopped because they stated they had a severe cold every time they took a vaccine injection.

Summary

Our study of the mass use of a cold vaccine made from bacterial organisms grown from upper respiratory tracts of people in the Houston area showed little difference whether a weak vaccine, a strong vaccine, or a saline placebo was used. Two hundred forty-six cases were studied in this series.

Another study, unpublished, used a stock autogenous vaccine made from selected cases and when given under careful observation showed improvement, with less upper respiratory infections and fewer colds.

A few cases were definitely made worse when taking the vaccine. Each time they had an injection they developed symptoms of an acute cold.

We feel the mass use of the vaccine is not practical. Its value in selected cases is yet to be proved, but there is a possibility it is effective where given under a better control than is possible where mass inoculations are carried out.

Further study of protein electrophoretic pattern may give a clue to cases where the vaccine could be successfully used.

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The Ear in Severe Malformations of the Head

A Discussion of the Formal and Causal Genetic Factors Involved

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This paper is the last of a series of eight dealing with malformations of the various parts of the ear. It is its purpose to review the ear changes in anencephaly and otocephaly, both severe malformations of the head in nonviable fetuses. An attempt will be made to analyze them according to the present state of our knowledge of the mechanics of head, and particularly ear, development. Finally, a short review of the ear changes in certain other double monsters will be given.

Anencephaly

Anencephaly (exencephaly, acrany) is a common malformation in man as well as in animals. It is characterized by defective development of the vault of the skull and the brain (for details see Ostertag). According to the degree of the osseous changes the cases are divided into two groups: (1) holoacrany, with extensive defect of the vault and absence of the posterior part of the occipital foramen, sometimes with even more extensive defects of the spine (craniorhachischisis), and (2) meroacrany, with the defect confined to the area adjacent to the sagittal suture and with the occipital foramen present (cranioschisis). The brain is either completely absent or represented by a fungous vascular mass, the so-called area cerebrovasculosa. In some instances the defect in the cranial vault is less extensive; rhombencephalon and mesencephalon are present, and the remnants of the prosencephalon protrude through the opening in the vault of the skull (pseudencephaly, hemicephaly). The development of the pituitary gland is frequently impaired, and this probably accounts for changes in other endocrine glands, particularly for the marked hypoplasia of the adrenal glands, commonly present in these cases.

The bones of the base of the skull show, as a rule, deformities of various degrees.

The most comprehensive study of the changes occurring in the temporal bones was carried out by Agazzi. A review of the reported findings shows a more or less rudimentary development of the squama; the petrous pyramid is shorter, thicker, and more solid than normal and occupies a more transversal position, with the apex standing higher than the base. The configuration of the internal auditory meatus is in most instances abnormal. Its entrance is narrow but its fundus very wide; the nerves within the meatus are in the majority of cases thin and the nerve fibers surrounded by numerous thin-walled blood vessels, so that pictures result reminiscent of the area cerebrovasculosa of the brain. No such vessels are, as a rule, seen within the modiolus, in the branches of the vestibular nerves, or in the facial nerve distal to the geniculate ganglion. The vestibular ganglion and, to a slighter extent, the geniculate ganglion are hypoplastic in many instances, and the ganglionic cells are scattered along the course of the nerves. The spiral ganglion is often hypoplastic, occasionally completely absent.

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Of the three parts of the ear, the inner ear shows the most marked changes. Under normal conditions the cochlea and the vestibule are situated with one behind the other in the direction of the long axis of the petrous pyramid. In anencephaly the two structures have undergone a rotation and have become approximated to each other whereby their respective longitudinal axes form an outwardly open obtuse angle of varying degree. The cochlea shows, as a rule, severer structural changes than the vestibule and the semicircular canals. Agazzi divides the cases according to the condition of the cochlea into the following three groups: 1. The cochlea is normal, except perhaps for slight defects in the axial skeleton of the upper part. 2. The number of cochlear turns is reduced to one to two and one-quarter turns, and the modiolus and the lamina spiralis ossea show a proportionate reduction in their development. The cochlear duct is not dilated. 3. The reduction in the length of the cochlear duct is accompanied by ectasy of the cochlear duct, and the findings resemble those of the so-called typus Mondini in hereditary deafness.

The organ of Corti is, as a rule, well developed. No definite relationship seems to exist between the condition of the nerves and the deformation of the cochlea.

In some instances rudimentary development or complete absence of one or two semicircular canals (Bauer, Buhl and Hubrich, Seligmann, Turkewitsch, Osawa) has been observed; otherwise, no abnormalities were found in the membranous parts, including the nerve endings, except perhaps a dilatation of the ductus and saccus endolymphaticus in some instances or a diffuse dilatation of the utricle and saccule together, with a very marked dilatation of the endolymphatic duct and sac (Nager).

The oval window might be smaller than normal and deformed or even completely absent.

Malformations of the oval window are always associated with deformities of the stapedial footplate. In a case reported by Agazzi (Case 10) the oval window was replaced by a small fissure filled with connective tissue. The stapedial footplate was found in its normal position; it showed normal configuration and was connected to the outer surface of the bony vestibular wall by a thin layer of connective tissue. Sometimes a partial synostosis was noticed between the footplate of the stapes and the window frame. The round window might also be absent or reduced to a narrow fissure. In a case reported by Frey the round-window membrane was covered on its outer surface, toward the round-window niche, by a thin bony lamella.

The changes in the two other parts of the ear are less characteristic. The size of the middle-ear cavity varies; it was absent in Alexander's case and very small in observations reported by Seligmann and by Turkewitsch. It was, however, larger than normal in other instances. A characteristic finding is numerous and irregularly shaped bony lamellae protruding from the walls into the lumen.

Of the ossicles, the stapes is often abnormal. The footplate is frequently very small, the crura elongated and approximated. Occasionally only one columella-like central bony column is noted, or one of the crura is absent with the other crus in its regular place. Sometimes lesser anomalies, such as small bony spicules or fibrous or osseous bridges between the crura, are observed. Complete absence of the stapes was seen by Alexander and by Turkewitsch. Anomalies of the outer ossicles are less common. Rudimentary development of the incus and malleus as well as excessive size or marked elongation was found in rare instances. Occasionally the handle of the malleus or the long process of the incus is shortened and the lenticular process absent.

The Eustachian tube is usually short, due to displacement of the tympanic cavity toward the midline; sometimes the tube is wide.

The facial nerve may show a slightly anomalous course across the petrous pyramid or within the middle ear.

The tympanic bone is displaced toward the midline, and the drum membrane occupies a more or less vertical position; its horizontal diameter approaches the frontal place (Eggert).

The external auditory meatus is usually normal, but was atretic in rare instances (Zingerle, Seligmann, Alexander). The auricle shows various degrees of deformities. If the external canal is atretic, it is severely deformed or even absent (Alexander).

Before discussing the significance of these findings, the results of the examination of the ear in three anencephalic featuses will be reported.

CASE 1.—Female infant, 37 cm. long and weighing 2610 gm.

History.—Mother 23 years old; para 3, gravida 3; two previous pregnancies uneventful. Family history negative. Delivery normal; 10 days before expected date. Child died shortly after birth.

Autopsy Findings.—Holoacrany, with extensive defect of the scalp, the cranial vault, and the posterior portion of the bony occipital foramen. From the eyebrows to the upper border of the atlas, the base of the skull was covered with a blood-red, gelatinous membrane. The petrous pyramids occupied a more transversal position than usual, with the apices standing higher than the bases. The nose was flattened, and the ears were pressed downward and forward though fairly well formed.

Among the other findings only hypoplasia of the adrenals will be mentioned.

Histologic Examination of the Temporal Bones (Right Side).—Vertical serial sections were studied.

External Ear: Membranous canal narrow, osseous canal short, but otherwise normal. Drum membrane thin, of normal configuration.

Eustachian Tube: Tube short but of normal structure.

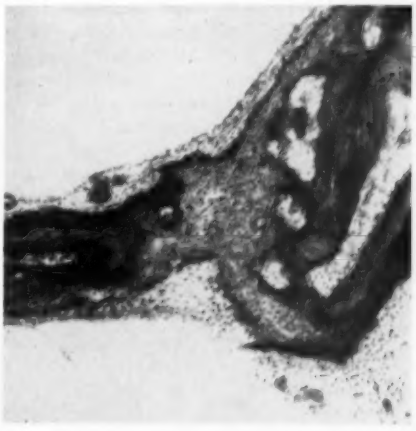
Middle Ear: The osseous tympanic cavity showed normal measurements, the lumen was well developed, wide, and the mucosal lining relatively thin. Epitympanum and antrum were well developed; the periantral cells were already pneumatized. Numerous bony crests protruded into the lumen of the hypotympanum. The oval-window niche was well developed, and the entrance into the round-window niche was transformed into a narrow channel; the round-window membrane itself showed normal configuration. Malleus, incus, incudomalleolar and incudostapedial joints, and styloid process were normal. The tensor tympani muscle was considerably thinner than normal. The stapedial crura were somewhat elongated and approximated; the footplate was irregularly shaped and thickened in its peripheral portions. The annular ligament was recognizable only in a portion of its anterosuperior circumference; otherwise, the footplate was fused with the window frame either by bone or by an intervening layer of cartilage (Fig. 1). The stapedial muscle was very thin. The facial nerve showed a normal course within the middle ear; it lay in a groove-like excavation of the medial tympanic wall and was covered on its outer surface by the middle-ear mucosa. The chorda tympani showed a normal course.

Inner Ear: The osseous labyrinthine capsule showed normal structure; the fissula ante fenestram was well developed; the fossula post fenestram was completely ossified.

The internal auditory meatus was of approximately normal configuration; its walls were lined with a thick layer of dural tissue. The nerve fibers within the meatus were thin, surrounded and separated from each other by a tissue containing numerous wide- thin-walled and well-filled blood vessels (Fig. 2). The vestibular ganglion was small, infiltrated with blood vessels. The facial nerve took its normal course to the geniculate ganglion; the latter was small but free from blood vessels.

Cochlea: The cochlea had one and one-half turns; the bony framework of the modiolus was thin; the septum between the upper half of the first and the second turn was absent. The cochlear

Fig. 1 (Case 1, right side).—Partial replacement of annular ligament by cartilage.



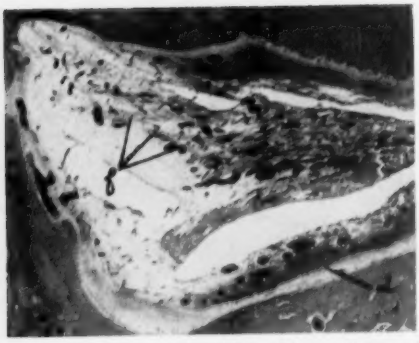


Fig. 2 (Case 1, right side).—Nerve fibers (*f*) near the fundus of the internal auditory meatus; *d*, thick layer of dural tissue.

duct described one and one-half turns and was of normal width. The organ of Corti showed marked postmortem changes, but was otherwise normal. Together with the stria vascularis it was present throughout the duct. The cells of the spiral ganglion were very markedly reduced in number; the nerve fibers going to the organ of Corti were less numerous and thin. The remainder of the spiral channel of the modiolus was filled with fine-fibrillar connective tissue with round and plasma cells and with some dilated blood vessels. The cochlear aqueduct was normal.

Vestibule and Semicircular Canals: The membranous structures, including the maculae, cristae, and endolymphatic duct and sac, were normal.

Histologic Examination of the Temporal Bones (Left Side).—Vertical serial sections were studied.

External Ear and Eustachian Tube: Same as on the right side.

Middle Ear: The bony trabecula in the hypotympanum was less prominent, the entrance to the round-window niche was wider, and the niche itself was filled with loose connective tissue. Pneumatization of the periantral cells had not progressed as far as on the right. The ossicles and the styloid process were essentially normal, the intratympanic muscles thin. The stapedial footplate showed normal configuration and structure; the annular ligament was incompletely developed and in numerous places replaced by cartilaginous and sometimes even osseous bridges between the footplate and the window frame. The facial nerve was of regular size and followed a normal course throughout the middle ear. The osseous canal was closed with the exception of a large dehiscence above the oval window.

A thin arterial blood vessel, measuring about 0.25 mm. in its diameter, branched off from the internal carotid artery, penetrating the floor of the tympanic cavity and entering the anterior part of the middle ear. It ran, covered by the mucosa, upward and backward over the posterior portion of the promontory, then left it and went upward between the crura of the stapes into the facial canal through the anteriormost portion of the dehiscence, a short distance behind the cochleariform process (Fig. 3). The vessel accompanied the facial nerve to the geniculate ganglion and then ramified. Because of the severe malformation of the skull an identification of its branches was impossible. The course of the chorda tympani was normal.

Inner Ear and Internal Auditory Meatus: Same findings as on the right side. The facial nerve did not go through the meatus on this side, but ran, covered with connective tissue, slightly behind the meatus over the upper circumference of the cochlea to the well-developed geniculate ganglion. It then entered the middle ear in a canal between the lateral surface of the cochlea and the root of the tegmen tympani. Its diameter was slightly smaller than normal.

The osseous labyrinthine capsule showed no abnormalities.

Cochlea: Same findings as on the right side.

Vestibule and Semicircular Canals: Normal.

Case 2.—Male infant, 47 cm. long and weighing 3120 gm.

History.—Mother 28 years old; para 1, gravida 2. Family history negative. Pregnancy uneventful. Delivery at term, normal. Child died 12 hours after birth.

Autopsy Findings.—Holoacrany with herniation of the rudimentary brain tissue through the defect. The eyes were prominent and the nose flat. The auricles were set low on the lateral surface of the face but were well formed; the neck was short.

Fig. 3 (Case 1, left side).—Stapedial artery (*a*) and stapes (*st*).

Among the other autopsy findings only hypoplasia of the adrenals will again be mentioned.

Histologic Examination of the Temporal Bones.—Oblique vertical sections were studied. Right and left sides showed identical findings.

External Auditory Meatus: Normal; drum of normal size and configuration.

Eustachian Tube: Short; structure normal.

Middle Ear: The tympanic cavity was of normal size; the lumen was narrow and extended from the tubar ostium backward along the inner surface of the drum just to the posterior pole of the latter. The rest of the cavity was filled with loose connective tissue. There were relatively few bony crests, protruding only into the hypotympanum and epitympanum. The round-window niche and the window itself were normal and so were the malleus, incus, incudomalleolar and incudostapedial joints, and styloid process. The tensor tympani and stapedial muscles were very thin. The head of the stapes showed normal configuration, and the stapedial tendon occupied its normal position. There was only one crus present, which, according to its location, represented the anterior crus of the normal stapes. It terminated distally in a rudimentary footplate (Fig. 4). The oval window was reduced to a fissure in the lateral vestibular wall, which was filled with cartilage. Ossification was in progress in the peripheral portions of this cartilage. The rudimentary stapedial footplate lay in the vicinity of this occluding plate. It consisted of enchondral bone; its medial surface was covered with a thin layer of cartilage and was connected to the vestibular wall by strands of dense connective tissue.

The course of the facial nerve was normal from the internal auditory meatus to the well-developed geniculate ganglion and from there throughout the middle ear. No osseous canal had been formed. The nerve itself was of normal size.

Above the stapes a small vessel branched off the artery, which accompanied the facial nerve downward from the geniculate ganglion; it went downward to the concavity of the stapedial crus, sending a small branch to its periosteum and terminating on the promontory (Fig. 5).

Fig. 5 (Case 2, left side).—Small blood vessel (*v*) going to rudimentary stapes (*st*) and promontory (*p*).

Internal Ear: The osseous labyrinthine capsule was normal; fissula ante and fossula post fenestram were both recognizable.

The internal auditory meatus had normal configuration; the contained nerves had a more or less normal appearance; there were only a few thin-walled blood vessels between the fibers. The cells of the vestibular ganglion were markedly reduced in number and replaced by small round cells.

Cochlea: Had two and one-half turns; the bony framework of the modiolus was thin and the

Fig. 4 (Case 2, right side).—Fissure (*f*) in labyrinthine wall, representing the oval window; *st*, rudimentary stapes.

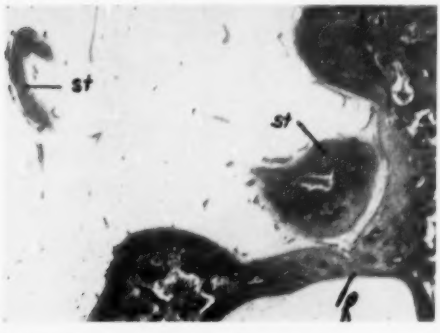


Fig. 6 (Case 2, left side).—Absence of septum between upper basal and the middle cochlear turn.

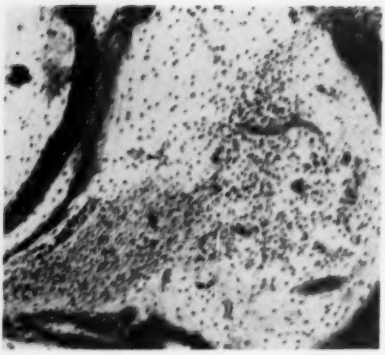


Fig. 7 (Case 2, left side).—Spiral ganglion; upper basal turn.

septum between the upper basal and the middle turn absent (Fig. 6). The cochlear duct and its contained structures appeared normal except for considerable postmortem changes. The cells of the spiral ganglion were greatly reduced in number and were small, often difficult to recognize. The spiral canal and the spiral lamina contained fine-fibrillar connective tissue with some thin nerve fibers and showed marked infiltration with lymphocytes and some plasma cells (Fig. 7). The cochlear aqueduct was well developed.

Vestibule and Semicircular Canals: All the contained structures, including the endolymphatic duct and sac, were normal.

Case 3.—Male infant, 46 cm. long and weighing 3340 gm.

History.—Mother 29 years old; para 2, gravida 2. Rh-negative; father Rh-positive. Two years before her last pregnancy she gave birth to twins, who, according to the history, are hemophiliacs; one mentally defective. Delivery uneventful, at term. Child died shortly after delivery.

Autopsy Findings.—Holoacrany with oval-shaped defect of the scalp and calvaria. The nose was flat and the eyes bulging; auricles normal. Among the other findings hypoplasia of the adrenals shall again be mentioned.

Histologic Examination of the Temporal Bones (Right Side).—Vertical serial sections were studied.

External Ear: No abnormalities.

Eustachian Tube: Wide; structure normal.

Middle Ear: Tympanic cavity and antrum of normal width. The lumen was confined to the lateral portion of the cavity, to the area adjacent to the drum, and extended just a short distance behind the latter; it was somewhat wider, as in Case 2. The remaining part of the cavity was filled with loose reticular connective tissue. No abnormal bony crests were seen in the hypotympanum or epitympanum, and the windows showed normal configuration. Malleus, incus, incudomalleolar and incudostapedial joints, and styloid process were normal; the intratympanic muscles were thin. Stapes was well formed, but the footplate was smaller than normal. The annular ligament was normal. Fissula ante and fossula post fenestram were present. The stapedial tendon occupied its normal position.

The course of the facial nerve from the internal auditory meatus to the well-developed geniculate ganglion and from there through the middle ear, as well as the course of the chorda tympani, was normal. The facial canal was closed in its upper part, but open in a part of its circumference below the oval window.

A thin artery, measuring about 0.25 mm. in diameter, left the internal carotid artery, ran upward, penetrating the anteriomedial portion of the floor of the middle ear, went to the surface of the promontory, and from there, covered by the mucosa, traveled upward and slightly backward until it reached the oval-window niche. It then left the promontory and went straight upward between the crura of the stapes to the facial nerve (Fig. 8); then with the nerve it traveled to the geniculate ganglion, where it ramified.

Inner Ear: The internal auditory meatus showed a slight widening of the fundus but otherwise was of normal configuration. The dural layer covering its surface was markedly thickened and contained numerous wide blood vessels. The facial nerve showed normal thickness; the fibers of the cochlear and vestibular portions of the eighth nerve were markedly reduced in number and size.

Fig. 8 (Case 3, right side).—Stapedial artery (*a*) and stapes (*st*).

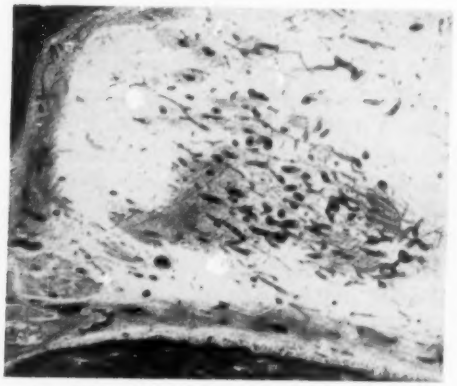


Fig. 9 (Case 3, right side).—Cochlear portion of eighth nerve.

They were surrounded and widely separated from each other by a tissue containing very numerous wide, thin-walled blood vessels which were engorged with blood (Fig. 9). The blood vessels became less numerous in the innermost portion of the fundus. No trace of Scarpa's ganglion could be found within the meatus.

The osseous labyrinthine capsule was normal.

Cochlea: Had only one and one-half turns. The bony framework of the axial portion was thin; the modiolus short, its upper portion absent. The spiral canal of the modiolus contained loose connective tissue with round cells and some plasma cells; no ganglionic cells could be positively identified. Occasional, thin nerve fibers were found in the spiral lamina. The bony septum between the upper basal and the middle turn was missing. The spiral lamina was present in the basal but absent in the middle turn. The cochlear duct had one and one-half turns; its width was normal. The organ of Corti was present in the basal turn only and showed marked postmortem changes. The stria vascularis was present in the entire duct. The cochlear aqueduct was normal.

Vestibule and Semicircular Canals: All the contained structures, including the endolymphatic duct and sac, were normal.

Histologic Examination of the Temporal Bones (Left Side).—Vertical serial sections were studied.

External and Middle Ear: Same findings as on the right side.

Internal Ear: Within the internal auditory meatus Scarpa's ganglion was present; small ganglionic cells well recognizable. Other findings as on the right side.

Cochlea: Had on the left side also one and one-half turns, but the axial bony framework was well formed; the configuration of the spiral lamina was normal. Cochlear duct was of normal width; organ of Corti and stria vascularis were present throughout the duct. The spiral canal on this side also was filled with reticular connective tissue with round and plasma cells. Only a very few ganglionic cells could be identified. Occasional thin nerve fibers were seen in the spiral lamina. The cochlear aqueduct was normal.

Vestibule and Semicircular Canals: All the contained structures were normal.

Summary of the Anatomical Findings.—The base of the skull showed, in all three cases, the characteristic configuration usually present in anencephaly and in consequence of it the already described changes in the position of the various parts of the temporal bone.

The auricle and the external auditory meatus were more or less normal in all three cases.

The middle ear was of normal size and configuration in Cases 2 and 3; on the right side of Case 1, and to a slighter extent on the left side, numerous bony crests were protruding into the hypotympanum and were narrowing the niche of the round window. The Eustachian tube, as usual in anencephaly, was short in all three cases, but otherwise normal. The middle-ear cavity was wide in Case 1 and poorly developed in Cases 2 and 3.

Of the ossicles, the malleus and incus were normal in all three cases; the stapes was essentially normal in Case 3, except for the smaller size of the footplate; in Case 1 it was of normal configuration on the left side, slightly abnormal on the right side. On both sides the footplate was ankylosed through the formation of cartilaginous and osseous bridges between the plate and the window frame, which replaced parts of the annular ligament. In Case 2 the oval window was on both sides reduced to a cartilaginous fissure in the bony vestibular wall; the posterior crus of the stapes was absent, and the footplate rudimentary and connected by short strands of connective tissue to the bony vestibular wall. The styloid process was normal in all three cases.

The intratympanic muscles were hypoplastic in all three cases.

The osseous facial canal was well formed on the left side and completely absent on the right side in Case 1, and it was absent on both sides in Case 2. In Case 3 it was well developed, except for a large dehiscence at the typical place above the window. The facial nerve itself and the geniculate ganglion were slightly hypoplastic in Case 1 and of normal size in the other two cases. The course of the nerve was normal, with the exception of that on the left side in Case 1. Here the nerve did not enter the internal auditory meatus, but ran slightly behind the meatus over the upper circumference of the cochlea to the geniculate ganglion; it then entered the middle ear in a canal between the lateral surface of the cochlea and the root of the tegmen tympani.

The chorda tympani was normal in all three cases.

The artery branching off from the internal carotid artery, entering the middle ear, and running over the promontory and then between the stapedial crura upward to the facial nerve and geniculate ganglion (which was present on the left side of Case 1 and on both sides of Case 3) represented a typical persistent stapedial artery.

The small vessel observed on both sides in Case 2, which branched off from the artery accompanying the facial nerve downward from the geniculate ganglion and which ran downward in the concavity of the anterior stapedial crus to the promontory, was not an anomaly but is regularly present in the newborn. It represents, most probably, according to Altmann (1934), a rudiment of the middle part of the main stem of the stapedial artery between the stapes and the point of origin of the supraorbital branch of the stapedial artery.

In the inner ear the configuration of the internal auditory meatus was normal in Cases 1 and 2; the fundus was slightly wider than normal in Case 3.

The cochlear and the vestibular portion of the eighth nerve were of approximately normal size in Case 2 and thin in Cases 1 and 3. In these two cases the nerve fibers were surrounded and widely separated from each other by a tissue containing very numerous and wide, thin-walled blood vessels reminiscent of the area cerebrovasculosa. In Case 2 these changes were much less pronounced. The vestibular ganglion was markedly hypoplastic in all instances and was completely absent within the internal auditory meatus on the right side of Case 3. The cells of the ganglion were reduced in number and size and to a large extent replaced by round cells and some plasma cells. The cochlea had one and one-half turns in Cases 1 and 3 and two and one-half turns in Case 2.

In all the cases, with the exception of the left side of Case 3, the axial skeleton showed slight defects in its upper portion. The cochlear duct was always of normal width, and the cochlear aqueduct was also normal. The organ of Corti showed marked postmortem changes, but apparently normal structure. It extended in all cases through the entire length of the cochlear duct, with the exception of the right side in Case 3, where it was present in the basal turn only and absent in the remaining upper half turn, whereas the stria vascularis was found throughout the one and one-half turns.

The spiral ganglion showed a considerable reduction in the number of ganglionic cells in all cases, with the exception of the right side of Case 3, where the ganglionic cells were completely absent. The spiral canal was filled with fine-fibrillar reticular tissue with round and plasma cells; in Case 1 it also contained some wide, thin-walled blood vessels. The nerve fibers were in all cases thin and greatly reduced in number, particularly so in Case 3.

The vestibular portion of the inner ear was free from anomalies in all three cases.

The labyrinthine capsule was of normal structure in all cases, with the exception of the replacement of the oval window by a cartilaginous fissure on both sides of Case 2.

Comment.—In order to understand the formal, as well as the causal, genesis of anencephaly, and of the associated ear mal-

formations, a short review of the present state of our knowledge of the mechanics of the development of the head in general, and of the ear region in particular, seems appropriate. It is mainly based on the results of experimental work in Amphibia.

Two main growth areas can be distinguished in the head region: 1. The prosencephalic, or rostral, area, which comprises the forebrain, midbrain, nose, and eyes, and which is underlaid by the anterior part of the prechordal plate. 2. The deuterencephalic, or caudal, area, which gives rise to rhombencephalon, labyrinth, and branchial gut; it is underlaid by the posterior part of the prechordal plate and chordamesoderm of the archenteric roof.

The central nervous system develops under the stimulus of this or these organizers, which induce not only the formation of the neural plate but also its subsequent transformation into the neural groove, tube, and eventually into the brain. In man by the seventh somite stage (about the 22nd day) the two lateral margins of the neural groove meet dorsally in the middle line in the region of the fourth to the sixth somite and fuse to form the neural tube. At this stage the cephalic two-thirds of the neural plate and tube, i. e., as far caudal as the fourth somite, constitute the primordium of the future brain.

In the absence of direct contact with the organizer, the ectoderm has only a feeble capacity to differentiate into nervous tissue. This could result in a corresponding failure of the plate to close, since closure depends on continued differential growth of the plate. When the neural plate remains unclosed, it is soon covered by an overgrowth of mesoderm.

A cause of abnormal contact between mesoderm and entoderm could be, according to Erskine, the presence of edema and cystic changes in the mesenchyme, which was noted by him in two anencephalic embryos of 14 and 18. mm. in length.

The teratogenetic termination period, i. e., the latest developmental stage at which the damaging factor must have been operative, is evidently the time of closure of the neural tube, which starts in embryos with seven somites (about 22 days old). The developmental disturbance could, of course, very well have started much earlier, but it is, at the present state of our knowledge, impossible to determine the actual moment of its onset.

The determination of the normal development by normal organizer action is apparently the result of the smooth interaction of a multitude of genes. The determination of the inherited malformations would in the last analysis be the result of abnormal genes, which cause abnormal organizer actions.

Human anencephaly must, however, not necessarily be the result of nonclosure of the neural tube. It could also develop as a result of rupture and partial resorption of an already closed tube. This can be concluded from the findings in pseudencephalic mice, which bear some close relationship to the findings in certain types of human anencephaly. The changes in mice are caused by a single recessive gene. The underlying disturbance is, according to Bonnevie, a disproportion in the growth of the medullary tube and the surrounding mesenchyme. A delay in the separation of the primary germ layers, particularly a delayed separation of the notochord from the ectoderm, which becomes manifest in embryos of seven to eight days, has shifted the balance of growth in favor of the medullary tube. In mouse embryos of about 10 days the spinal cord has already become too big in proportion to the surrounding mesenchyme and forms irregular curvatures in the neck region and elsewhere. Rapid growth of the brain with increase of the intracranial pressure soon leads to a rupture of the brain with outward prolapse.

Bivetti describes the findings in six anencephalic human embryos of 14-30 mm. in length with varying degrees of anencephaly and assumes from them that there is a developmental mechanism similar to that Bonnevie found for the pseudencephalic

mice. Patten assumes from findings in human embryos that local overgrowth of the neural plate in early developmental stages might be a factor in the genesis of craniochisis or rhachischisis. This overgrowth might either interfere with the closure of the tube or lead to rupture of the already closed tube.

The changes in pseudencephalic mice are due to a single recessive gene; identical changes have also occurred in mice as a consequence of imbalanced chromosome combinations (translocations) in the offspring of male mice which were exposed to x-rays (Snell and Picken).

These observations lend support to the assumption still held by some geneticists that human anencephaly is exclusively the result of abnormal gene action. This view (monomeric recessive heredity) was, for instance, held as late as 1950, expressed by Polman, and Böök and Rayner (lethal genotypical factor).

Lately there has, however, been a growing realization of the fact that a large but yet undetermined proportion of the malformations is due not to genetic but to environmental factors (see Warkany, 1947). According to Grebe (1953), there are hereditary, as well as nonhereditary, forms of anencephaly in man. Both types seem to be equally frequent.

Rübsaamen and Leder, and particularly Werthemann, in a recent review of the subject, enumerate a multitude of factors which could influence the developing embryo, among them lack of oxygen, infectious diseases of the mother during the first trimester of pregnancy, vitamin deficiencies, radiation, etc.

Kaven, Russell, and Hicks, for instance, produced typical malformations of the central nervous system in mice and rats with single low doses of x-rays. The patterns of the malformations ranged from anencephaly through hydrocephalus to various kinds of microcephaly and hydrocephalus. Radiation of rats in the earliest somite stage, on the ninth day of pregnancy, produced anencephaly together with severe defects of the head. The malformations of the central nervous system caused by ionizing radiation, or by other noxious agents, are most probably the result of damage to the primitive neuroblasts and spongioblasts (Hicks). The ensuing patterns of malformations are determined by two factors, the character of the agent and the time of injury.

Through the courtesy of Dr. Josef Warkany (Children's Hospital, Cincinnati) I had the opportunity to examine the temporal bones of a rat whose mother had been radiated with 1120 r on the 15th day of gestation. The mother was killed on the 22nd day of pregnancy and 12 young were obtained (see Warkany and Schraffenberger). These young appeared externally very abnormal, since they had short upper and lower jaws. All had cleft palates. The hands and feet were small and underdeveloped, the tails short and curly. The ribs showed abnormalities; the long bones were essentially normal.

RAT 2A 1605a.—Serial sections were prepared for histological examination of the head.

Brain.—Severely malformed, with absence of the corpus callosum and hydrocephalus.

Ears.—Identical findings on both sides.

External Ears.—Normal.

Middle Ear.—Eustachian tube well developed; tympanic space narrow, slit-like; the remaining part of the tympanic cavity filled with loose reticular connective tissue. Round window normal; oval window absent. Malleus well developed, completely cartilaginous; incus rudimentary, reduced to a small piece of cartilage representing the body and articulating with the head of the malleus. Stapes, styloid process, and stapedius and tensor tympani muscles absent. Facial nerve normal, giving off the chorda tympani, which ran forward over the medial surface of the handle of the hammer. The stapedial artery entered the middle-ear space just behind the point of exit of the facial nerve. It then ran forward and upward along the medial wall of the middle ear and eventually penetrated in a short canal the base of the skull.

Inner Ear.—Capsule still completely cartilaginous; oval window and cochlear aqueduct absent.

The place of the cochlear duct was taken by a slightly curved cystic sac (Fig. 10); the saccule and utricle were likewise very wide and communicated with each other through a wide opening;

Fig. 10 (Rat 2A 1605a, right side).—Cystic sac replacing the cochlear duct (*c*) and communicating through a wide opening with the saccule (*s*).

endolymphatic duct and sac were absent. The cochlear cyst communicated with the saccule through a wide opening; the border between the saccule and utricle was marked by a low circular fold projecting into the lumen. The perilymphatic cisterna had become almost completely obliterated by the dilated saccule and utricle. Both maculae were present. The cochlear cyst showed, on its posteromedial circumference, a band-like epithelial thickening which revealed the typical structure of the organ of Corti of a newborn rat; tectorial membrane was well formed and stria vascularis also recognizable. The membranous canals, including the cristae, were normal. The eighth nerve supplied the maculae and cristae, as well as the organ of Corti, with fibers.

Briefly summarized the following anomalies were present:

Inner ear: absence of endolymphatic duct and sac, dilatation of utricle and saccule, transformation of cochlear duct into a cystic sac.

Middle ear: absence of stapes, stapedial muscle, and styloid process; rudimentary development of incus; absence of tensor tympani muscle.

The just-described findings resemble those described by me (1955) in two rats, the mother of which had received Trypan blue injections in two-week intervals before pregnancy and one injection on the 1st, 14th, and the 15th day of pregnancy. There was, however, a herniation of the dilated cochlear duct through the internal auditory meatus in one of them and a severer malformation of the middle ear in both of them.

Similar but still more marked inner-ear changes, without anomalies in the middle ear, had previously been described by Hertwig in the offspring of male mice which had received an x-ray dosage of 1000 r (x-ray-mutated circler mice).

The question naturally arises whether in the just-described observation the changes in the ears could be the result of the radiation on the 15th day of embryonic life, or if they represent coincidental findings.

The absence of the endolymphatic duct and sac points to a teratogenetic termination period for the inner-ear malformation of about the 10th day, 5 days before the radiation. On the 15th day the gross differentiation of the membranous labyrinth, including the endolymphatic duct and sac, is almost complete. In the middle ear all the ossicles are present and in the transition from the precartilaginous to the cartilaginous stage. Under these circumstances, it does not seem very probable that the ear changes are the result of the radiation. It seems much more likely that they represent coincidental findings and that they are not related to the experimentally produced skeletal changes.

The just-discussed case showed clearly how important a careful evaluation of all the findings in a given observation is in order to avoid conclusions which appear at first sight rather obvious but which, nevertheless, under closer and critical scrutiny, prove erroneous.

The next problem requiring discussion is the developmental mechanics of the ear. It has been shown in Amphibia that the otic vesicles develop under the inductive influence of the head mesoderm, the hindbrain, and possibly also the neural crest; the mesoderm seems to be the primary inductive factor (for a review of the problem see Hörstadius and also Yntema, 1955).

Mesodermal induction is believed to occur mainly during late gastrular and early

neurular stages; neural induction, during late neurular and early head-process stages. Paired mesodermal inductors can be localized in the early gastrula; the mesodermal activation actually starts with the migration of the notochord mesoderm beneath the prospective ear ectoderm. It grows in intensity until the neural folds start to form, then diminishes, and eventually becomes completely extinct.

Mesodermal activation is followed by activation localized in the neural folds. The ear ectoderm comes to lie adjacent to the folds during neurulation, and so is exposed to neural activation. This influence is most effective during the process of fusion of the neural folds and then decreases in intensity. The further normal differentiation of the otic vesicle is thought by many authors to be contingent on the presence and intactness of the medulla oblongata. There is, however, evidence that the medulla is not necessary for late differentiation. If the hindbrain is removed at the auditory placode stage a normal labyrinth, nevertheless, forms.

Another essential factor seems to be the intactness of the endolymphatic appendage. Otocysts which fail to develop a ductus or saccus endolymphaticus may swell and remain vesicular. The presence of an intact endolymphatic appendage is, however, no guarantee of normal differentiation of the otocysts; it may be present in cases with cystic endolymphatic labyrinth.

The determination of the otocyst for the development of a normal labyrinth does, therefore, not occur at one particular stage but occurs gradually. It is not completed until a relatively late developmental stage, and it probably involves several different factors in succession. The relative importance of the two types of induction for the normal development varies among different groups of Amphibia. Mesodermal induction alone results in imperfect, small ears. By the time the auditory placode has formed, the rudiment is able to develop into a normal labyrinth without further activation by neural crest or tube.

There are strong indications that the development and differentiation of the otic vesicles in mammals is governed by essentially the same factors as in amphibians. The findings in the already mentioned x-ray-mutated circler mice (Hertwig) and in tissue cultures of isolated otocysts of rats (Lawrence and Merchant) seem to favor this assumption.

In amphibians, the epithelium of the otic capsule exerts a formative influence on the surrounding embryonic mesenchyme and induces the formation of a labyrinthine capsule. After extirpation of the otic capsule in frogs, not only the cartilaginous labyrinthine capsule but also the medial parts of the sound-conducting mechanism fail to develop; the outer parts remain normal. Similarly in chickens, extirpation of the otocyst results in complete absence of the labyrinthine capsule and in improper development of the footplate of the columella; all the other parts of the middle ear remain normal.

These findings are in agreement with the fact that in man the stapedial footplate has a dual origin. The stapedial rim, the vestibular surface, the basal perichondrium, and the annular ligament are derived from the lamina stapedialis of the labyrinthine capsule, the remainder of the footplate from the second visceral bar (Cauldwell and Anson, 1942).

The development of the tubotympanic pouch and its derivatives is evidently independent of that of the inner ear, and vice versa.

Since anomalies of the stapes, of a derivative of the second visceral arch, occur commonly in anencephaly, it will also be pointed out that the visceral arches develop under the inductive influence of the rhombencephalic head organizer. Judging from the findings, particularly in Amphibia, the major portion of the cartilages and bones of the visceral skeleton seems to originate from the cells of the neural crest (see Hörstadius). The latter takes its origin from the ectoderm, becomes separated from it at an early

developmental stage (before closure of the neural tube), and extends on either side of the midline in a craniocaudal direction in the angle between the neural material and the epidermis. Cellular proliferation within the neural crest leads to ingrowth of cell masses in a ventral direction, into the visceral arches. A participation of the mesectoderm of the neural crest in the formation of the visceral skeleton can also be assumed to take place in man, according to the findings of Bartelmez and Evans.

The dorsal portions of the neural crest participate, together with the lateral placodes of the ectoderm, in the formation of the sensory ganglia of the head (for details see Hörstadius and also Altmann, 1950).

When applying the just-outlined concepts to the analysis of the anencephaly, of the ear changes and their mutual relationship, one must keep the following facts in mind. If nonclosure of the neural tube is the cause of anencephaly, the teratogenetic termination period of this malformation would be at an early stage of embryonic development, around the seventh somite stage (2 mm., or about 22 days), a stage at which the otic placodes have just become prominent structures. Should the anencephaly, however, be due to rupture of an already closed neural tube, the teratogenetic termination period would be at a later stage and the development of the inner ear would have progressed much further.

For the inner-ear malformations in anencephaly which are confined to the cochlea, the teratogenetic termination period varies mainly according to the number of cochlear turns present. One turn is formed in 18-20 mm. embryos (about 40 days), one and one-half turns in 30 mm. embryos (about 50 days), and two and one-half turns in 40-50 mm. embryos (60-70 days).

Since the number of cochlear turns varies from one to two and one-half turns, the teratogenetic termination period would thus vary from 6-10 weeks (18-50 mm. embryos). Such a late date explains, according to Agazzi, why the changes in the membranous labyrinth are, as a rule, confined to the cochlear duct; at the time when the teratogenetic influences were active, the cochlear duct was just developing, the other parts, however, having already developed and no longer subject to change.

In the cases with rudimentary development, or absence, of the semicircular canals the teratogenetic termination period would be at the time of the anlage of the two pouches from which the canals develop (embryos of 8-9 mm., or about five weeks).

The frequent anomalies of the stapes and the less common anomalies of the other ossicles would require a teratogenetic termination period at the time of the first appearance of the anlagen of the ossicles as condensations in the mesenchyme of the second and first branchial arches. This would occur in approximately 7 mm. embryos (32 days), since the first anlage of the stapes becomes visible at this stage.

The changes in the cochlear and vestibular ganglia and the less pronounced changes in the geniculate ganglion are not necessarily due solely to a defective anlage but, at least partly, are also due to degenerative processes within the ganglia and nerves during embryonic life. This would explain the presence of great numbers of round and plasma cells within the ganglia.

The first common anlage of the three ganglia, the acousticofacial ganglion, is clearly discernible in embryos of 30 somites (4-5 mm., 28 days). It is derived mainly from the otic vesicle and the neural crest. Shortly afterwards, in 5-6 mm. embryos, the facial ganglion becomes completely separated from the acoustic ganglion; the latter retains its close relationship to the otocyst. Nerve fibers grow soon after the first appearance of the ganglion toward the brain. The motor fibers of the facial nerve already extend in 6.9 mm. embryos from the neural tube toward the mesenchyme of the second branchial arch; they are joined by the peripheral fibers of the geniculate ganglion. In 7 mm. embryos the acoustic ganglion has become partially subdivided into a superior

and an inferior portion, each of which develops its own group of peripheral nerve branches. In 9 mm. embryos (about 35 days) the spiral ganglion makes its first appearance as an accumulation of ganglionic cells and one sees fibers growing from it toward the brain. When the formation of the cochlear duct takes place, the cell mass lies in the concavity of the first anlage, and it retains this relationship in later stages.

In view of these data, the teratogenetic termination period of the anomalies of the ganglia and nerves could also very well occur during the 8-9 mm. stage.

Summarizing one can, therefore, say that the assumption of a damaging influence present not later than at the 7-8 mm. stage could satisfactorily explain all the known ear anomalies in anencephaly. If one is solely guided by the cochlear changes and fails to consider the other findings, one would arrive at a much later, but most probably incorrect, date.

In regard to the mutual relationship between the malformations of the central nervous system and those of the ears, one should remember that not only the nervous system but also the otic vesicles, as well as the visceral arches, develop under the inductive influence of the same structure, of the material of the prechordal plate. It might also be of interest to point out again that, at least in Amphibia, the cells of the neural crest give origin not only to the major part of the acousticofacial ganglion but also of the visceral skeleton. It is, therefore, understandable that disturbances in the head organizer can cause anomalies in the central nervous system as well as in all parts of the ear. The neural activation of the otic vesicles which follows the mesodermal activation is, at least in Amphibia, most effective during the process of fusion of the neural folds, up to the time the otic placodes have formed. The determination of the malformation of the otic vesicles, as well as of the ossicles, must, therefore, also have taken place at a considerably earlier stage than even the earliest time considered on the basis of the morphological findings as the teratogenetic termination period for these malformations.

Otocephaly

Otocephaly (synotia) comprises a group of malformations which occur only rarely in man but oftener in certain animals such as sheep, cattle, dogs, guinea pigs, mice, and rabbits. (See Blanc, Josephy, H. Marx, Wright and Wagner, Pires de Lima, and others.)

Otocephaly is often associated with varying degrees of cyclopia (see Wright and Wagner).

According to the severity of the malformation, the following types can be distinguished (Blanc):

In ageniocephaly, or agnathia, the type most frequently observed in man, the mandible is highly rudimentary or sometimes even completely absent; the oral cavity is rudimentary and the hypopharynx occluded; the external ears are shifted to the neck and markedly approximated, and the middle-ear cavities approach each other and are in some instances already fused. Maxillae, palatal bones, and sphenoids are rudimentary. Cranium and brain are normal.

In sphenocephaly also the mandible is highly rudimentary or absent and the oral cavity rudimentary. Zygomatic bones, temporal bones, pterygoid processes, and maxillae are shifted in a ventrocaudal direction and approximated; the alveolar processes are bent toward the midline, and the auricles are either already fused or still separate. The external auditory meatuses are, accordingly, either fused into a single canal or still bipartite. The tympanic cavity presents various degrees of fusion.

In agnathocephaly the mandible as well as the oral cavity are absent. Zygomatic bones and arches are shifted more caudoventrally, and the eyes show a similar displacement. There is further approximation of the maxillae, with partial fusion of the alveolar processes. Auricles are approximated or united at their base, squamosal bones approximated

or fused at their base, and tympanic cavities approximated or fused. Cranium and brain are still well formed.

In strophocephaly the eyes lie in a common orbit on the undersurface of the head. Zygomatic bones and arches are absent, the maxillae highly rudimentary, and the squamosal bones fused below the sphenoid. Auricles and tympanic cavities are approximated or fused. The nose is severely malformed and represented by a process situated above the eyes (proboscis). Cranium and brain are still normal.

In edocephaly the eyes become fused, the nose is either represented by a proboscis above the eyes or is completely absent, and maxillae are below the sphenoid. The auricles are deformed, approximated, and lie below the fused eyes, at the border between the head and neck. Their long axes stand almost horizontal, and the auricles are slightly fused at the approximated ends. Tympanic cavities are approximated or fused. The brain is malformed and cystic.

In severer degrees of cyclopia (opocephaly, spherocephaly) the nose is absent and the eyes rudimentary. Auricles, tympanic cavities, and Eustachian tubes are fused.

In triocephaly, the severest degree of these malformations, the face is completely absent, except for the ears, which are united at their base. The petrous pyramids are approximated or fused in the midline.

In guinea pigs, otocephaly appears sporadically in certain inbred strains. All the already mentioned types of malformations are found, and, according to the severity, 12 degrees were distinguished by Wright and Eaton, and Wright and Wagner, starting with hypoplasia of the mandible and ending with full-fledged triocephaly. The last two authors give the most comprehensive analysis of the findings in otocephaly in general and its relationship to cyclopia.

A review of the ear findings shows essentially identical conditions in the less severe forms of the malformations (see Blanc, Josephy, Marx).

The auricles are present and show, as a rule, aside from the shift in their position, only slight changes, not exceeding a moderate degree of microtia (Otto, Guerdan). Marked shift to the midline might be accompanied by fusion of the lobules (Hoffmann). The external auditory meatuses are either separate, converging, or already fused.

The middle-ear changes are also essentially determined by the tendency to fusion. The annuli tympanici might be approximated, connected by a bony rod, or fused into one bone containing a single drum membrane for both external auditory canals. The tympanic cavities might be united and are sometimes considerably dilated. The ossicles are either normal or deformed and partially defective or completely absent (Kuse). In several instances, such as in Arnold's case, the heads or the handles of the hammers were connected by a small bony rod, which was regarded as rudimentary cartilage of Meckel. The fenestrae are sometimes narrowed or closed with connective tissue or bone.

The Eustachian tubes are sometimes fused, shortened, or even completely absent.

The inner ears are, as a rule, normal.

In triocephaly, which so far has been observed only in animals, the whole skull is highly rudimentary; the temporal bones are always present, although markedly deformed. Aside from several macroscopically examined cases, all in sheep, a similar case was recently studied histologically by Millen and Davies.

In a sheep fetus of 82 mm. in length, with an approximate age of seven weeks' gestation, the head was rounded and lacked the normal projection of the snout region. The pinnae were approximated, and there was no indication of the nose, eyes, or mouth.

The pharynx ended at the level of the attachment of the hyoid cartilages to the auditory capsules. It was continuous cranially with a very narrow diverticulum, which was considered to represent the fused right and left tubotympanic cavities. Cranially the diverticulum opened onto the anterior surface of the head, a little above the pinnae. Below the opening, there was a well-

marked meatal plug which did not quite reach the tubotympanic diverticulum.

The cartilaginous auditory capsules were approximated, and the anterior part of the base of the skull with the ethmoid was missing.

Meckel's cartilages were reduced in size and fused into a single cartilage lying dorsal to the tubotympanic diverticulum. On its caudal end this cartilage was connected laterally to the right auditory capsule and articulated anteriorly with the right cartilage of Reichert. Membranous ossification around the cartilage suggested an attempt to form a mandible (Fig. 11).

The cranial ends of Reichert's cartilages were attached on both sides to the labyrinthine capsule. They then passed in a caudoventral direction toward the midline, almost meeting in front of the tubotympanic diverticulum, and then turned caudally to connect with the hyoid apparatus in a normal manner.

The only recognizable ossicle was the left stapes; it lay deep to Reichert's cartilage and was connected to it by a cartilaginous bar. It had two normal crura encircling a stapedial artery but lacked a footplate. The oval window was absent. On the right side, oval window, stapes, and stapedial artery were absent.

The facial nerves emerged from foramina in the auditory capsules, turned around Reichert's cartilages, and united in the midline. Branches were given off to the muscles associated with the cartilages; several ganglionic masses were found in relation to the facial nerves and their branches.

The inner ears, including the auditory nerves, were normal.

Forebrain and midbrain were absent, but the hindbrain and its associated nerves were present.

The trigeminal ganglion and nerves were of approximately normal size and distribution.

The authors explain the findings with a primary defect in the foregut, with subsequent failure in the induction of the anterior part of the neural plate and of the associated neural crest.

The conditions described in this sheep embryo seem to demonstrate a close developmental relationship between the nervous system and the neural crest, on the one hand, and the visceral arch mesoderm, on the other. The findings at the cartilaginous base of the skull, especially in the region of the trabecular cartilages, strikingly resembled the results of neural crest excision in Amphibia (Hörstadius).

The absence of the stapedial footplate and of the oval window is again explained by the dual origin of the stapes, from Reichert's cartilage and from the lamina stapedialis of the otic capsule.

Agnathia, and to a certain extent the entire group of otocephalic malformations, is distinguished from the other types of malformations of the first visceral arch by three characteristic features: a constant, severe malformation of the mandible; a relatively mild involvement of the ear; a combination of the two with severe malformations of the foregut derivatives—mouth, pharynx, and esophagus. The degree of the latter malformations runs parallel to that of the mandible.

The great variety of the malformations of the first branchial arch, ranging from dysostosis mandibulofacialis, or the Treacher-Collins syndrome, to agnathia, is due to the relatively independent behavior of the various component parts of the first branchial arch and cleft toward the causative agents, whatever they might be. Lehmann, in a recent review and analysis of the known anatomical findings and of the results of experiments in Amphibia, comes to the conclusion that the otocephalic malformations are the result of damage to the deuterencephalic, or caudal, head organizer. Since the nervous system and the inner ear are only very rarely affected, one must assume that the malformation develops at a time when the induction of the rhombencephalon and of the otic vesicle has been more or less complete but not later than that. The determination of the extent of the malformation takes place at a still earlier date, at the time of the determination of the various organ anlagen within the previously not too highly differentiated blastema field (so-called segregation).

Since damage to the prosencephalic head organizer leads to arrhinencephaly and cyclopia, the severest degrees of otocephaly are evidently due to damage not only to the caudal but also to the rostral head organizer.

Double Monsters

The ear changes occurring in certain forms of double monsters are of limited

interest and will only be discussed briefly. For more detailed information concerning double monsters in general the reader is referred to the classical work of Schwalbe and for information concerning the ear to the review of Marx.

Ear changes are found in monsters with ventral fusion of the two individual parts above the umbilicus (cephalothoracopagus, prosopothoracopagus) and in those with varying degrees of anterior duplication of the body axis (duplicitas anterior, diprosopus, dicephalus).

In the so-called monosymmetric form of the cephalothoracopagus (Schwalbe) one of the "secondary anterior surfaces" shows a regular face, while on the opposite surface the medial parts of the face are missing. The ears are approximated or fused.

According to the size of the median defect, Marx distinguished three types of ear changes.

In Type 1, the mildest degree of synotia, the auricles are approximated and touch each other. The external meatuses converge in the depth, are separated from each other by a septum, or partially united. The middle-ear spaces are separated and the labyrinths normal.

In Type 2, the auricles are fused in the lower part. The meatuses are separate or united; the middle-ear spaces are united into one space. Both mallei and incudes are fused; the labyrinths are essentially normal.

In Type 3, the auricles are fused and more or less hypoplastic. There is only one meatus which ends blindly. The middle-ear space is hypoplastic or absent, and the labyrinths are malformed. The two ears on the nondefective surface are either normal or also show anomalies (Alexander, 1899).

The prosopothoracopagus is characterized by ventral union of the chests and oblique lateral union of the heads. One finds on the anterior surface of the united heads a more or less complete duplication of the face and on the posterior surface various degrees of synotia with various degrees of union of the inner parts.

In anterior duplications one finds, according to the degree of duplication, all the transitional stages between complete absence of the medial ears in slight degrees of duplication and normal formation of the medial ears in marked degrees.

In the so-called asymmetric double monsters one individual part is considerably less developed than the other fully developed individual part. The less developed part is called the parasite, the other one the autosite (Schwalbe).

In cases of cephalothoracopagus parasiticus the auricles are often relatively well developed even when the other parts of the head are missing.

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Direct Adenoidectomy

III. Results

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Results of direct adenoidectomy in 36 cases of conduction hearing impairment in children were reported by L. K. Guggenheim [1] 13 years ago. No similar report has appeared in the subsequent literature, although the surgical principle of complete adenoidectomy under direct vision in such cases has gained wide acceptance. Since the value of any treatment for the alleviation of deafness is measured only by results, it would seem no less worth while to record the effects of adenoid surgery performed by various techniques than the results of fenestrations, stapedolyses, x-ray, radium treatment, etc.

The purpose of the present communication is to report a series of 30 cases. Twenty-one of these are pediatric. Upon 14 of these I performed the first tonsillectomy and adenoidectomy (Group I cases). Seven are cases of adenoidectomy revision (Group II cases). Unlike L. K. Guggenheim's material, this series also includes some adults and adolescents, nine in number, upon whom either primary or secondary surgery was performed (Group III cases).

Such data require that preoperative and postoperative hearing tests be recorded in all hard of hearing cases. Indeed, L. K. Guggenheim impressed upon me the desirability of performing audiometric screening upon all candidates for T. and A. irrespective of the complaint of impaired hearing. This procedure has revealed a surprising number of children with subclinical hearing losses which might well have proved to be of later importance. It is noteworthy that audiometric defects may closely approach the critical 30 db. level without bringing the child's problem to the attention of the parents. Yet the margin of safety, the reserve, is nearly exhausted. Many such children, if neglected, will subsequently develop clinical losses due to progressive, irreversible tympanal fibrosis.

Routine audiometric screening no doubt imposes a burden of added work upon the routine of a busy office. Yet, it seems no less logical to screen these children, who are already patients, than to screen school children generally. Quite commonly even children with obvious hearing defects are not tested prior to T. and A., for it is assumed that the operation, by whatever technique it is performed, will do everything possible to alleviate the existing defect.

Since each surgeon uses the technique most satisfactory in his own hands, no one is in a favorable position to produce comparative statistics of results from different methods. It is therefore urged that each operator interested in advancing the specialty study his own clinical material and compare his results with those of others.

My experience agrees with L. K. Guggenheim's that complete adenoidectomy with tubal dilatation will restore normal hearing in the overwhelming majority of cases of conduction deafness due to Eustachian-tube obstruction from hypertrophied and infected adenoids. This improvement occurs in from one to three months following surgery. Routine audiometric follow-up is done not sooner than one month postoperatively.

L. K. Guggenheim [1] reported that such ideal results were obtainable, for the most part, in children up to 13 years of age. Beyond that age, presumably as a result of hormonal changes, there was believed to

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be a progressively increasing tendency for adult scar tissue to be deposited in the tympanic cavities, with production of an irreversible fibrosis. I have found, nevertheless, that surprising results are sometimes obtained in adolescents and even adults as a result of complete adenoidectomy. While one can be less certain of the outcome, the effort is still distinctly worth while from the point of view of hearing.

It has been my experience, in common with others, that adequate adenoidectomy markedly diminishes the frequency of otitis media. Nonetheless, we are occasionally distressed when our adenoidectomy cases experience a later attack. One must remember that even in the absence of any tubal obstruction or other "anatomic predisposing factor" other influences can work to the patient's disadvantage. A particularly virulent bacterial invasion, injudicious blowing of the nose during an acute upper respiratory infection, allergy, or hypogammaglobulinemia may be responsible.

Guild [2] has pointed out the desirability of long-term follow-up in such cases as those reported herein, preferably for periods up to two years. A number of factors make such long-term study exceedingly difficult in this age of increased social mobility. Not the least of these is the "minor" nature of the surgery (from a lay viewpoint).

I can attest that even shorter term studies (up to a half year) are generally lacking. Although the results reported herein are not long-term according to the standards of Guild, it is my impression from the limited number of cases which I have been able to follow for longer periods, that occasional attacks of otitis media which follow complete adenoidectomy but are properly treated, do not permanently impair the good results already achieved in restoring hearing. In consequence, it seems probable that the results reported herein represent permanent improvements.

The Factor of Unresorbed Mesenchyme

L. K. Guggenheim [1,3] had observed that a few cases of conduction impairment, failing to achieve full restoration of hearing after complete adenoidectomy, improved satisfactorily after subsequent dilatation of the tubal orifices with the olives of von Gyergyai. The rationale of this procedure has been extensively discussed elsewhere.[4]

I have tried to follow a suggestion of Goodhill [5] to perform unilateral dilatation upon children with bilaterally symmetric audiometric defects in order to compare the

TABLE 1.—*Summary of Results of Unilateral Tubal Dilatation as Compared with Those of Bilateral Dilatation*

Case No.	Dilatation	Result
	Group I Cases	
1	Bilat.	Slightly asymmetric, not significant
2	Bilat.	Symmetric
3	Right side only	Very slight asymmetry favoring right ear, not believed significant
4	Bilat.	Symmetric
5	Right side only	Symmetric
6	Right side only	Symmetric
8	Bilat.	Essentially symmetric
11	Bilat.	Symmetric
12	Bilat.	Symmetric
13	Bilat.	Symmetric except for 125 d.v.
14	Right side only	Definitely asymmetric, favoring right ear
	Group II Cases	
15, 16, 18, 19, 21	Bilat.	Symmetric
	Group III Cases	
22	Bilat.	Asymmetric (chronic fibrosis unilaterally, case not relevant)
24	Right side only	Symmetric (defect originally minimal and on right side only)
29	Right side only	Symmetric (defect originally minimal and on right side only)

TABLE 2.—*Data on Thirty Cases*

Case	Age	Sex	P.I.	Previous Rx or Surgery	Operation	Clinical Follow-Up
1	5	M	D_c,O,R,C,A,S		T&A,$^{++}$,W	Mild mumps, encephalitis 1 wk. P.O. No allergy treatment. S persists.
2	8	M	D_c,Sp,O$^+$,T		T&A^{++}	Speech defect disappeared.
3	8	F	D_c,T,H		T&A^{++}(rt. only)	H improved to normal.
4	4½	F	D_c,E(rt.),O(lt.),T		T&A^{++}	
5	4	F	D_c,T		T&A^{++}(rt. only)	
6	6	F	O,D_c,T,Ad,R		T&A^{++}(rt. only)	
7	6	F	S,T,D_c		T&A,W	
8	8	M	D_m,T		T&A^{++}	Alleviation of D_c.
9	8	M	S,D_c,T		(1) T&A,$^+$,W (2) Antrum windows, plastic repair of nasopharynx; acrylic obturator worn 3 mo. 6 wk. Cortisone	(1) Cicatricial obliteration of nasopharynx. S persists. (2) S cured. Good patent nasopharynx. Little change in D_c.
10	7	F	D_c,T		T&A	
11	5	M	D_c,Ad,R,S,T,H		T&A,$^{++}$,W	S cured. H improved to normal.
12	8	M	T,A^{++},O$^+$,D_c	X-ray Rx at 5	T&A,$^{++}$,W	One severe O$^+$ (rt.) 2 mo. P.O. Ultimate hearing result unimpaired.
13	8	F	O,T,R,D_c		T&A^{++}	
14	7	F	D_c,Ad,Sp,R		(1) T&A^{++} (rt. only) (2) 2 mo. later lt. tubal dilat.	
15	5	M	D_c,C,A$^+$,O$^+$,E(lt.),R	T&A at 3	A^{++}	
16	6½	M	D_c,Ad,O,C,S, mother otosclerotic	T&A at 4	(1) T&A^{++} (2) Bilat. myringotomy; penicillin	(1) Bilat. E,P.O. S persisted because overlooked. (2) Uneventful recovery.
17	9	M	O.M.P.C. (rt.) since age 1; D_c,Ad,H	T&A at 7	Modified radical mastoidectomy and A$^+$ in one session	Small, dry perforation; of rt. M.T. (previously polyp). H improved. Nasal breathing restored. Slight audiometric change.
18	5	F	S,Ad,D_c(lt.)	T&A at 4	A^{++},W	S cured.
19	6	F	O$^+$(rt.), D_c,Ad	T&A at 2	A^{++}	
20	9	M	O$^+$(lt.) D_c(lt.),T,Ad (Leptothrix)	T&A at 7	T&A^{++}	No more O$^+$ (followed 2 yr.).
21	8	M	O$^+$(acute mastoiditis, bilaterally),D_c,Ad	T&A at 6	Bilat. simple mastoidectomies in 2 sessions; later A^{++}	
22	16	M	D_m(lt.) from old simple mastoidectomy for mastoiditis with "coma," Ad, H	Lt. simple mastoidectomy at 6 T&A at 7	A^{++}(lt. only)	H improved. Nasal breathing better.
23	25	F	O.M.P.C., bilat. from childhood, with bilat. aural polyps,D_c,Ad		T&A$^+$(local)	Ears healed with closure of perforations. Nasal breathing improved.
24	16	M	D_c (rt.),S,T		T&A^{++}(rt. only),W	Sinusitis cured.
25	25	M	O.M.P.C. (rt.) from childhood	Tonsillectomy at 20	T&A$^+$	Dry right ear with posterior marginal perforation.
26	12	F	D_c	T&A at 9	T&A$^+$	1 wk. P.O. acute S,O (lt.) dc cured.
27	14	M	D_c (rt.)	T&A at 11	T&A$^+$	1 wk. P.O. acute S,O (rt.) dc cured.
28	25	F	Chr. adenoiditis, bronchitis, fever, tinnitus, R	T&A at 4	Mysteclin. T&A$^+$	Tinnitus, bronchitis, P.N.D., fever, cured.
29	13	M	Ad,A,H	Elimination diet; antihistaminics	T&A^{++}(rt. only)	Nasal breathing restored; allergy improved.
30	25	F	O.M.P.C., bilat. from childhood; acute mastoiditis (rt.)		Rt. simple mastoidectomy; later T&A$^+$(loc.)	Both ears dry with large inferior marginal perforations.

The following is a key to the abbreviations used in Tables 2, 3, 4, and 5:

Deafness or hearing impairment	D
Subscript c=conduction type	
Subscript m=mixed type	
Subscript n=nerve type	
Allergy (nasal)	A
(nasal and asthma)	A$^+$
(nasal and asthma and eczema)	A^{++}
Adenoid obstruction (facies)	Ad
Frequent colds	C
Tonsilitis	T
Serous effusion of tympanum	E
Acute otitis media, nonpurulent	O
Acute otitis media, purulent	O$^+$
Chronic otitis media, purulent	O.M.P.C.
Sinusitis	S
Speech defect	Sp
Retracted drums	R
Below par general health status	H

Procedures

T&A$^+$	includes resection of lateral curtains.
T&A^{++}	includes also dilatation of Eustachian orifices.
W	designates antrum irrigations (washes).
T&A$^{\prime\prime}$	in Class II cases and some Class III cases are obviously revisions, but no special designation is necessary to indicate this.
A$^+$ or A^{++}	indicates adenoidectomy revision when no tonsil tissue was present. Tonsil tissue was removed whenever present.

result with that of the undilated ear. Under the restrictive limitations imposed by such an experiment, the relevant cases are necessarily few in number. Applying the utmost skepticism and considering as significant only markedly asymmetric results in unilaterally dilated cases, one case alone in the present series qualifies (Case 14). Since the incidence of unresorbed mesenchyme in the ears of children generally is not known, it is very difficult to assess the statistical significance of this case. The over-all results of this admittedly fragmentary experiment are summarized in Table 1. Fuller details are given in the analysis of the cases which follows.

Analysis of Cases

Cases have been divided into three categories for convenience of analysis:

Group I comprises 14 cases in which I performed the first T. and A.

Group II comprises 7 cases in which secondary surgery (T. and A. revision) was performed.*

Group III comprises 9 cases of adolescents or adults upon whom either primary or secondary surgery was performed.

Operations in which no curtain dissection or tubal dilatation was performed are referred to simply as "T and A." The

* With one exception (Case 9), no revision of a Group I case occurred in this series. The exception was the child who developed a cicatricial obliteration of the nasopharynx. This boy's younger sister, who had had a T. and A. performed by me without lateral curtain dissection or tubal dilatation, also developed considerable scar tissue in the nasopharynx—evidently a familial trait. The girl had no hearing loss and no sinusitis, and her scarring was discovered as the result of an opportunity afforded by repair of a laceration of her soft palate from a fall on a stick. Her hearing was not affected by the scarring. It is believed that the brother's sinusitis may have been responsible for the greater scarring which he suffered. His condition was discovered when, because of persistence of the sinusitis and increased hearing loss, I performed bilateral intranasal antrostomies and undertook what I anticipated to be simply an adenoidectomy revision. The obliterating scar tissue was removed by careful scalpel dissection. No skin grafting was done. An acrylic obturator was worn for three months, during half of which period the boy received 100 mg. of cortisone daily. An excellent result was achieved, including cure of the sinusitis. Hearing improved to the original preoperative level—a very moderate defect.

TABLE 3.—*Hearing Results in Group I Cases*

Case	128	256	512	1024	2048	2896	4096	5792	8192	11,584	End-Results
							Right Ears				
1	15	20	35	25	25	20	25	25	15	—	Speech range normal; 12-20 db. residual loss above and below
2	20	15	40	45	30	40	50	40	55	45	Completely normal
3	20	15	30	20	30	30	40	25	25	10	15 db. residual loss for 125,250
4	15	10	20	20	15	10	25	30	35	—	20-25 db. residual loss for 125,250, 500,1000 15 db. residual loss for 2000
5	20	20	20	30	15	10	20	10	25	45	Completely normal
6	25	35	40	30	15	15*	15*	20	10*	10*	Completely normal
7	10	25	20	10	15*	5*	5*	10*	5*	10*	15 db. residual loss for 500
8	5*	5*	5*	10	5	20	20	25	10	—	Dx D_m, alleviation of D_s
9	10	5	5	5	5	0*	0*	0*	0*	0*	Hearing essentially unchanged
10	5	5	5	5	25	10*	15*	25	35	10*	12 db. residual for 125, 250,500
11	20	25	20	25	20	15	15	20	40	25	Completely normal
12	10	*	*	*	*	*	*	*	10	15	Completely normal
13	10	15	10	15	10*	10*	20	20	10*	10*	20 db. residual for 125 (1 mo. postop.)
14	25	40	25	30	15	15	5*	10	10	0*	Completely normal
Av.	15.7	19.1	22.5	26.0	18.2	20.0	26.9	22.7	26.0	28.0	
							Left Ears				
1	40	25	25	25	30	25	30	25	30	—	Normal except 20 db. residual loss for 12,000
2	15	20	20	30	40	25	35	20	50	45	Completely normal
3	5	10	15	15	20	20	10	15	25	10	15 db. residual loss for 125, 250
4	0	0	15	20	30	10	35	25	35	—	Same as right ear (see above)
5	10	25	15	20	15	20	15	25	40	25	Completely normal
6	20	25	20	20	10*	10*	5*	15*	10*	15	Completely normal
7	25	25	25	20	0*	0*	0*	0*	0*	10*	15 db. residual loss for 125
8	30	20	10	10	15	15	15	10	20	—	Dx D_m, alleviation of D_s
9	5	10	5	5	0	0	0*	0*	0*	0*	Hearing essentially unchanged
10	0*	0*	0*	0*	15*	20	15	15	5*	15	Completely normal
11	35	25	35	30	25	30	25	35	20	20	Completely normal
12	15	20	15	30	20	35	35	30	20	25	Completely normal
13	10	5	5*	10*	25	30	25	5*	0*	20	Completely normal
14	10	30	30	25	0*	10*	10*	10*	35	25	Completely normal (after secondary tubal dilatation)
Av.	16.9	18.5	19.2	20.8	22.0	20.9	24.0	22.2	30.5	22.2	

For the meaning of abbreviations used, see footnote to Table 2.

TABLE 4.—*Hearing Results in Group II Cases*

Case	128	256	512	1024	2048	2896	4096	5792	8192	11,584	End-Results
						Right Ears					
15	25	30	40	25	25	—	40	45	35	25	Completely normal
16	30	20	25	35	30	40	30	35	35	20	Completely normal
17	10	5	10	5	10	5	25	15	0*	0	O.M.P.C. cured, little over-all change in hearing.
18	−5*	5*	0*	5*	5*	0*	15	5*	15*	20	Completely normal
19	—	20	20	35	25	—	30	25	30	30	20 db. residual for 125,250,500; 15 db. for 1000
20	*	*	*	*	*	*	*	*	*	*	
21	20	10	15	20	*	*	*	*	*	*	Completely normal
Av.	21.2	17.0	22.0	24.0	22.5	22.5	28.0	30.0	33.3	19.0	
						Left Ears					
15	20	15	30	35	40	—	25	25	25	15	Completely normal
16	15	15	15	20	15	25	25	40	25	10	Completely normal
17	10	10	0*	10	5*	0*	15*	15*	20	5	Normal except 20 db. residual for 125 and 11,584
18	25	15	15	10	15*	15*	20	5*	15*	15	Completely normal
19	10	15	5	5*	5*	*	15	15	30	30	20 db. residual for 125,250,500
20	10	5	10	15	15	15	25	25	15	35	Completely normal
21	*	*	*	*	*	*	*	*	10	15	Completely normal
Av.	15.0	12.5	15.0	18.0	23.3	20.0	22.0	26.2	20.8	17.8	

For the meaning of abbreviations used, see footnote to Table 2.

additional performance of curtain dissection is designated by "T&A.+" Tubal dilatation, which was carried out only when lateral curtains, if any, were first resected, is herein designated by "T&A.++" In a limited number of cases of symmetric or near-symmetric hearing losses, dilatation was carried out on one side (right) only, and this information has been appended as a parenthesis to the designation "T&A.++"

For brevity, past histories and clinical findings have been presented in tabular form. End-results, aside from hearing, are included in Table 2.

Audiometric improvements are also presented in tabular form with comments on the end-result as to hearing (and other

TABLE 5.—*Hearing Results in Group III Cases*

Case	128	256	512	1024	2048	2896	4096	5792	8192	11,584	End-Results
						Right Ears					
22	15	*	*	*	*	*	*	*	*	*	Completely normal.
23	40	40	40	45	20	30	10	20	20	40	Normal except 20 db. residuals for 4000, 6000 (possibly D_a). O.M.P.C. since childhood-polyp. Ear completely dry; perforation healed.
24	15	20	5*	5*	5*	10*	20*	15	30	25	Completely normal.
25	20	20	10	5	0	10	10	20	15	15	O.M.P.C. since childhood. Residual D_a but air-bone gap closed.
26	20	20	15	15	10*	15	5*	10	0*	0*	Completely normal.
27	15	15	25	25	25	20	15	35	20	20	Completely normal.
28	10	20	15	10*	*	*	*	*	*	*	Completely normal.
29	15	20	15	*	*	*	*	*	*	*	Completely normal.
30	10	25	45	25	5	5	10	25	20	20	O.M.P.C. since childhood. Acute mastoiditis; simple mastoidectomy before T&A†. Dry ears. Residual loss 20 db. for 125; 15 db. for 1000, 2000, 3000.
Av.	17.7	22.5	23.6	23.0	12.5	16.0	11.2	20.8	21.0	24.0	
						Left Ears					
22	20	5	−5	5	−5	−10	5	20	30	30	Old simple mastoidectomy. Nerve involvement (coma). D_a alleviated.
23	15	15	10	15	10	20	20	15	10	5	Normal except 20 db. residual for 8000; 25 db. for 11,584 (possibly D_a). O.M.P.C. since childhood-polyp. Ear completely dry; perforation healed.
24	10	15	10*	10*	*	*	*	*	*	*	Completely normal.
25	20	25	25	5	5	2	2	15	5	2	Residual D_a but air-bone gap closed except for 15 db. at 1000.
26	15	15	10	*	*	*	*	*	*	*	Completely normal.
27	5	10	15	10*	*	*	*	*	*	*	Completely normal.
28	20	15	15	10*	*	*	*	*	*	*	Completely normal.
29	*	*	*	*	*	*	*	*	*	*	
30	5	15	20	15	25	20	25	15	20	15	O.M.P.C. since childhood. Dry ear. Residual loss 20 db. for 125; 15 db. for 250.
Av.	13.8	14.4	12.8	10.0	8.8	8.0	10.4	16.3	16.3	10.4	

For the meaning of abbreviations used, see footnote to Table 2.

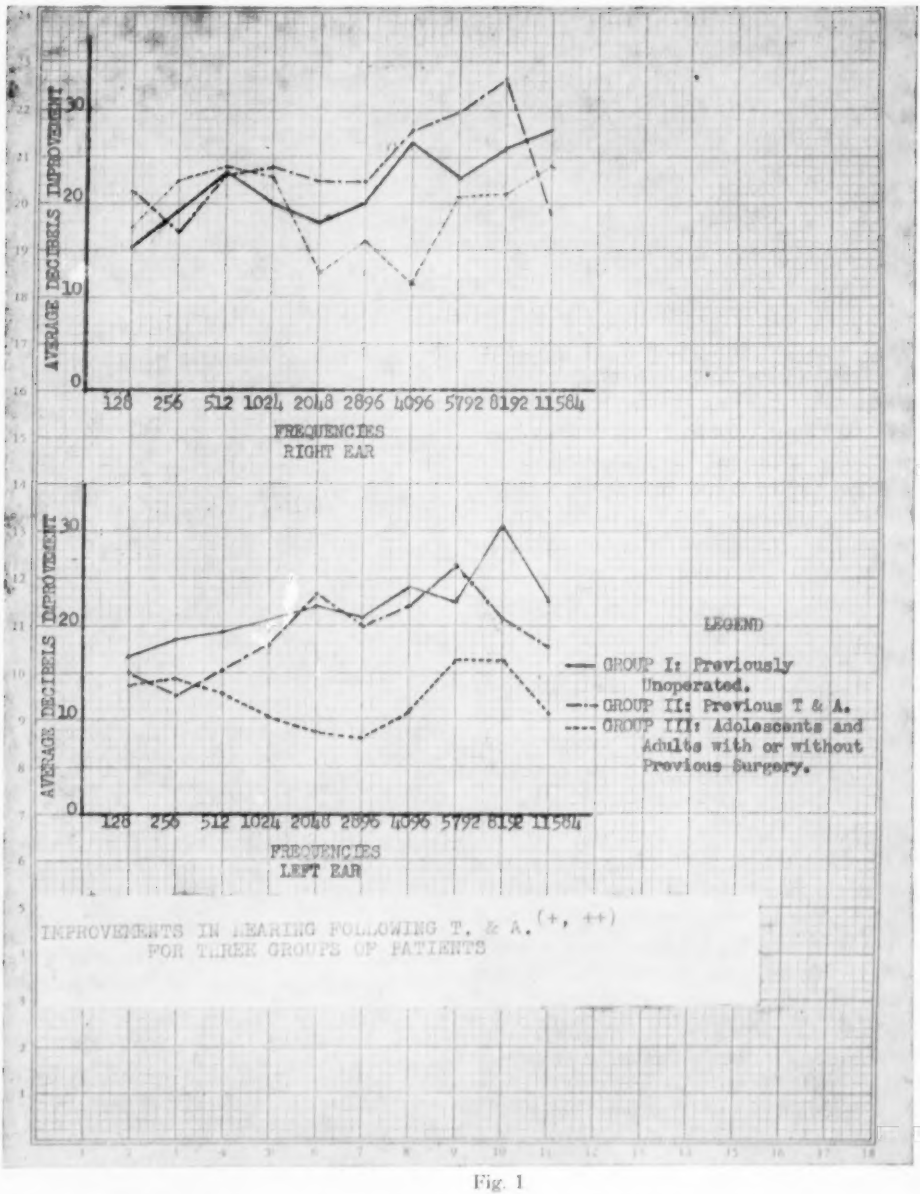


Fig. 1

factors involving the ear alone). Groups I, II, and III have been tabulated separately (Tables 3, 4, and 5) and average improvements for each frequency calculated for each group. Where initial hearing loss does not exceed 10 db., improvements (if any) are not included in the averaging calculation. (Figures for the improvement may or may not be entered in the appropriate spaces, but all such spaces are designated by an asterisk.)

The average decibel improvements at the various frequencies for each ear have been

TABLE 6.—*Incidence of Normal Initial Hearing Measurements in Three Groups of Patients*

	Right Ears Asterisks			Left Ears Asterisks		
	Total	Per Indiv.	% of All Measurements	Total	Per Indiv.	% of All Measurements
Group I	34	2.43	24.3	29	2.07	20.7
Group II	25	3.6	35.7	20	2.86	28.6
Group III	32	3.56	35.6	39	4.33	43.3

* Dr. George Rhodes, of the Department of Vocational Rehabilitation, Winter Veterans Hospital, helped with the statistical and graphic aspects of this study.

graphically plotted for all three groups (Fig. 1).

An impression, gained from glancing at the curves, that no significant difference exists between Groups I and II has been rigorously confirmed by application of the standard *t*-test.* From this result the conclusion seems warranted that adenoidectomy revision is capable of salvaging cases in which primary surgery has failed to give satisfactory improvement, with end-results comparable to those of successful primary surgery.

The gradual upward slope of the two curves is also noteworthy, indicating that greater improvement is to be expected with rise in frequency.

The curves for Group III cases would appear, at first glance, to indicate that lesser improvements are to be expected in this age group, as pointed out by L. K. Guggenheim.[1] Several considerations prevent one from regarding the present results as a fair test of this assertion. Group III, including cases of both primary and secondary surgery, does not constitute a class comparable with Groups I and II from the standpoint of research design. Comparison of Table 5 with tables 3 and 4 reveals many more spaces with asterisks in the former for left ears (Table 6).

Even a casual inspection reveals many more minimal losses in Group III, a factor which limits the magnitude of improvements necessary to obtain even ideal results. On the other hand, Group III includes more instances of chronic, irreversible major temporal bone disease in which no appreciable hearing improvements could be expected from adenoid surgery or, indeed, any other treatment modality. No doubt these characteristics of Group III are inevitable considering the tendency of passing years to heal wounds and to add scars. Ideally, a large group of adolescent and

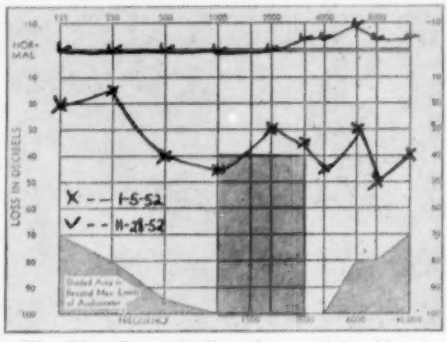
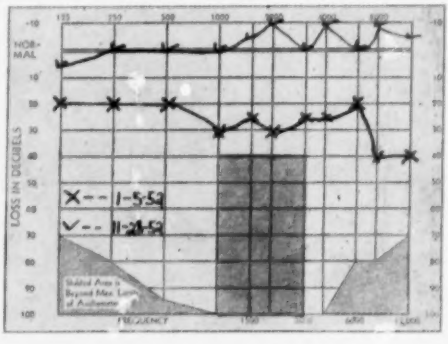


Fig. 2A.—Group I, Case 2 (sample): Air-conduction audiogram for right ear.

Fig. 2B.—Group I, Case 2 (sample): Air-conduction audiogram for left ear.



* Dr. George Rhodes, of the Department of Vocational Rehabilitation, Winter Veterans Hospital, helped with the statistical and graphic aspects of this study.

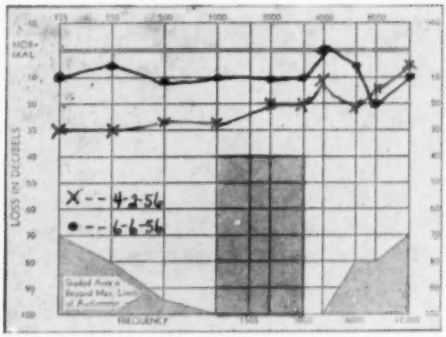


Fig. 3A.—Group I, Case 14 (Goodhill experiment): T. and A.* with right tubal dilatation performed on April 9, 1956. Two months' postoperative result is shown. Air-conduction audiogram for right ear.

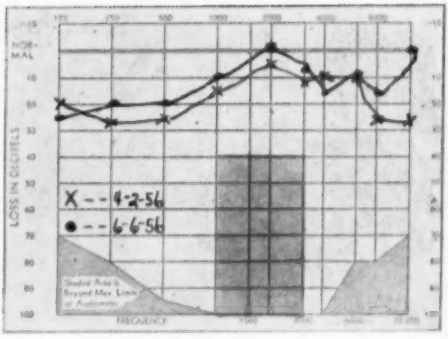


Fig. 3B.—Group I, Case 14 (Goodhill experiment): Air-conduction audiogram for left ear.

Fig. 3C.—Group I, Case 14 (Goodhill experiment): Slight further gain as shown on follow-up (by Dr. Philip Meltzer, of Boston).

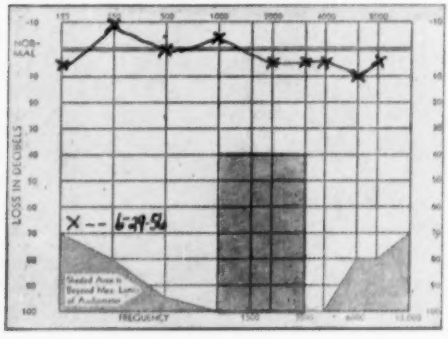


Fig. 3D.—Group I, Case 14 (Goodhill experiment): Final ideal result 21 days after delayed tubal dilatation as recorded by Dr. Meltzer.

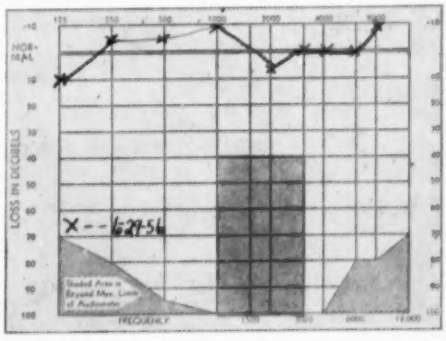


Fig. 4A.—Group II, Case 16 (sample): Air-conduction audiogram for right ear.

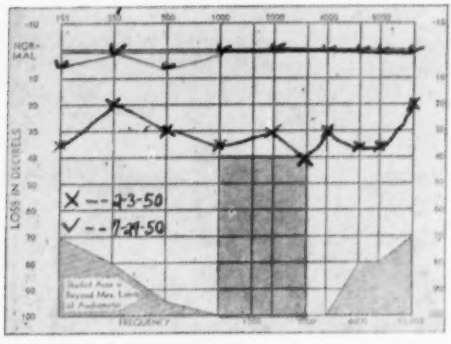
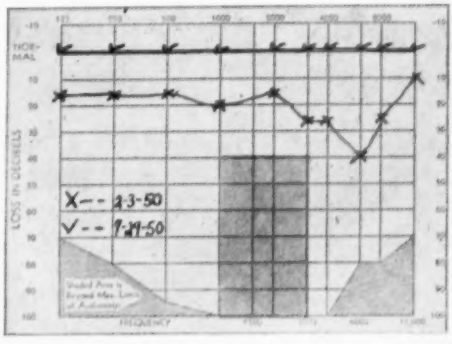


Fig. 4B.—Group II, Case 16 (sample): Air-conduction audiogram for left ear.



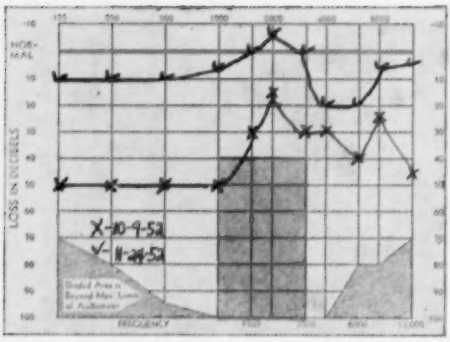


Fig. 5A.—Group III, Case 23: A 25-year-old housewife with bilateral O. M. P. C. since childhood presenting granulations protruding through large central perforations bilaterally. Both ears became dry, perforations healed, and hearing was restored to practically normal. Air-conduction audiogram for right ear.

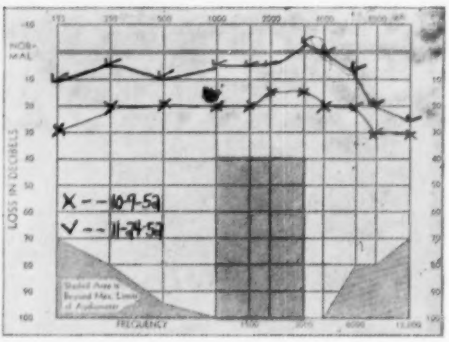


Fig. 5B.—Group III, Case 23: Air-conduction audiogram for left ear.

adult cases should be studied in which cases of primary and revision surgery would be separated, as in Groups I and II, and cases of irreversible temporal bone disease eliminated except for the one crucial condition which L. K. Guggenheim visualized as mitigating the results in patients past age 13—insidious tympanal fibrosis in the absence of major suppurative disease of the middle ear or mastoid.

In consequence, no real comparison is to be drawn between the hearing results in Group III cases and the essentially identical results in Groups I and II. The figures for hearing improvements, as well as the alleviation of suppurative conditions, indicate, suffice it to say, that adenoid surgery in the adolescent and adult has much to offer.

Individual audiograms of four cases are presented in full. Three are merely samples of their respective groups, one (Case 14) the crucial case for the effectiveness of tubal dilatation (Goodhill experiment).

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The Dry Physiologically Perfected Fenestra Nov-Ovalis Technique

A Technique for Predictable Restoration and Conservation of Serviceable Hearing in Clinical Otosclerosis

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Ever since Bárány [1] first observed that opening of the external semicircular canal improved hearing in clinical otosclerosis, the pioneers interested in the evolutionary development of the fenestration operation realized that to make the procedure entirely successful they would have to find definitely prescribed means for preventing postoperative osteogenetic closure of the newly created fenestra if the improved hearing was to be maintained permanently. It became obvious, also, that prevention of postoperative inflammatory reaction of the vestibular and the cochlear labyrinth was a necessity, since such inflammation, when severe enough, could not only interfere with maintenance of the immediate hearing improvement obtained by opening the perilymph space of the vestibular labyrinth, but might also result in still further impairment of hearing.

After experimental trial with monkeys and the adoption of changes in my techniques, I succeeded in devising and prescribing a logical means for consistently preventing the osteogenetic closure of the newly created fenestra in the surgical dome of the vestibule. I had observed in both monkey and man that when the portion of the tympanomeatal flap covering the fenestra gap became invaginated, by accident, sufficiently to cover and adhere to the fenestra rim osteogenetic closure of the fenestra was physiologically impeded. Therefore, I deliberately invaginated the portion of the flap facing the fenestra gap in order to encourage and facilitate its adhesion to the fenestra rim, thereby consistently preventing osteogenetic closure of the fenestra [2] (Fig. 1). Thus, the incidence of osteogenetic closure in my use of the fenestra nov-ovalis procedure was reduced to less than 2%. The final solution of this major problem was gratifying; a great stumbling block to successful fenestration surgery had been overcome.

There still remained, however, the other problem I have mentioned which had to be solved before the fenestration operation could reach the state of perfection necessary for obtaining maximum functional results with a minimum of operative after-effects. This was prevention of postoperative inflammatory reaction of the endolymphatic vestibular and cochlear labyrinths. The solution to this postoperative problem was vital for two main reasons: first, because of the discomfort to the patient from the prolonged severity of the vertigo which resulted from the postoperative inflammation; second, and most important, because occasionally the serous labyrinthitis was severe enough to irreparably damage the cochlear nerve function which had existed preoperatively.

Otologists everywhere interested in the improvement and perfection of the fenestration operation were trying to determine the cause of the postoperative inflammatory reaction of the endolymphatic labyrinth, always a postoperative occurrence, varying only in the degree of its severity, as judged by the postfenestration clinical picture. George Shambaugh, Jr.[3] suggested logically that

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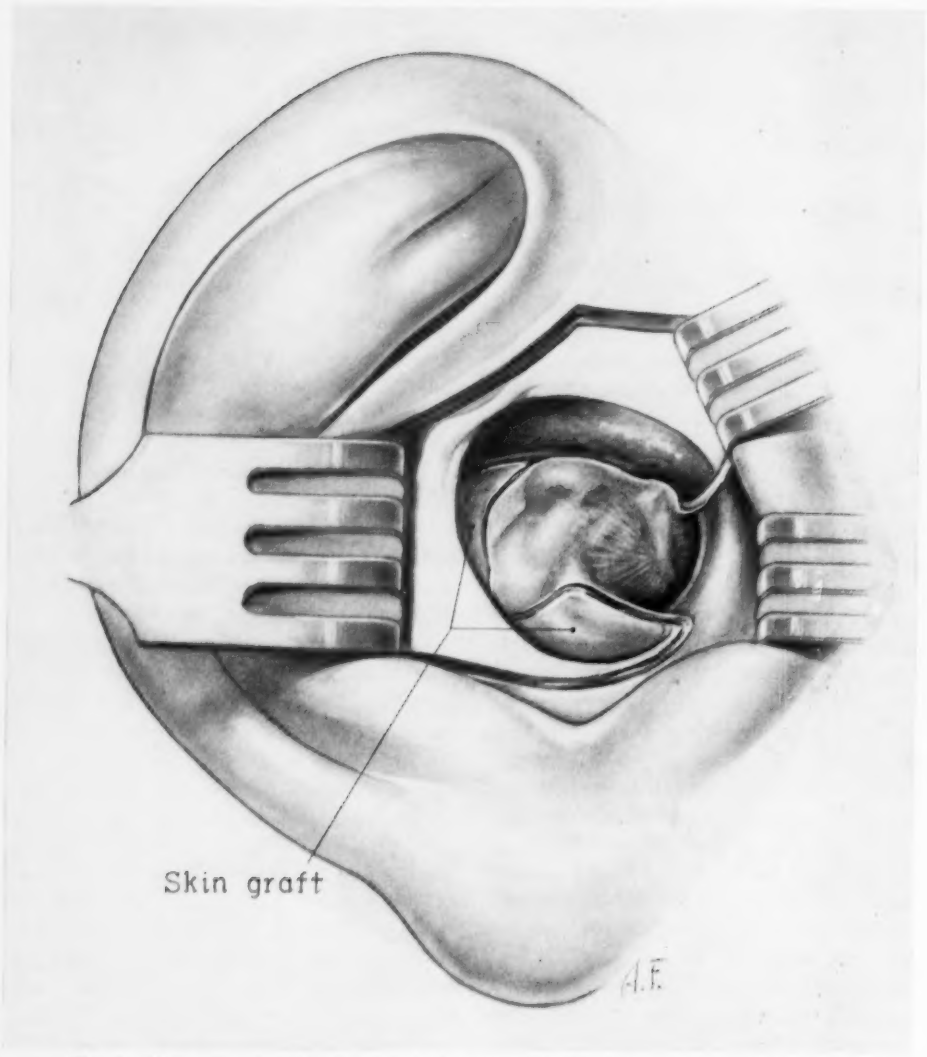


Fig. 1.—The prevention of osteogenesis is accomplished by invagination of the flexible, thin, untrimmed periosteum-lined portion of the flap nearest the tympanic membrane into the perilymph space sufficiently to contact the fenestra rim.

blood entering the perilymph space during the fenestration operation can cause postoperative serous labyrinthitis of a greater or lesser degree, varying according to the amount of blood entering the perilymph space. He proved, experimentally, in the monkey, that by permitting quantitatively different amounts of blood to enter the open perilymph space of the fenestra in the external semicircular canal several varied degrees of postoperative labyrinthitis could be produced. Nevertheless, the question still to be answered was "Will the avoidance of the technical error of permitting blood to enter into or start within the perilymph space prevent the onset of postoperative serous labyrinthitis?".

In my own research into the etiology of postoperative serous labyrinthitis I employed the process of elimination in order to de-

termine whether bleeding into or within the perilymph space is the sole cause of the postoperative inflammatory reaction of the endolymphatic labyrinth after the performance of the fenestration operation. Careful observation of the sources of origin of free blood in the perilymph space led me to devise techniques for preventing both the bleeding into and the bleeding within this area. My observations and the steps in my procedure are as follows:

1. Since failure to control all bleeding points within the exenterated mastoid cavity before opening the perilymph space of the newly created fenestra permits blood to enter the perilymph space, I avoided opening the perilymph space until every one of the bleeding points within the exenterated mastoid cavity had been controlled with the polishing burr.

2. Having observed that bleeding can be started within the perilymph space by injury to the perilymphatic trabeculae in attempts to retrieve and remove (by means of picks or irrigation) bone chips lost within the perilymph space, a common occurrence when employing the pulverization technique for creating the fenestra. (Fig. 2), I developed the cupola technique for opening the perilymph space.[4] Instead of pulverizing the bony labyrinthine capsule and pushing the bone chips into the perilymph space, the intact bony cupola is lifted and removed in one piece, in a direction away from the perilymph space (Figs. 3, 4, and 5). By employing my cupola technique for opening the perilymph space the initiation of bleeding within the perilymph space can be avoided.

3. Because bleeding into the perilymph space can take place from the inner surface of the meatal portion of the tympanomeatal flap when the surgically trimmed and periosteum-freed portion thereof is used to cover and seal the newly created fenestra, I tailored and placed the tympanomeatal flap so that I could always use the untrimmed, thin, periosteum-lined portion nearest the tympanic membrane to reach and cover the newly created fenestra, within the surgical dome of the vestibule, close to the facial nerve canal.

Fig. 2.—The pulverization technique for creating the fenestra nov-ovalis makes the necessity for irrigation in the presence of the opened perilymph space unavoidable.

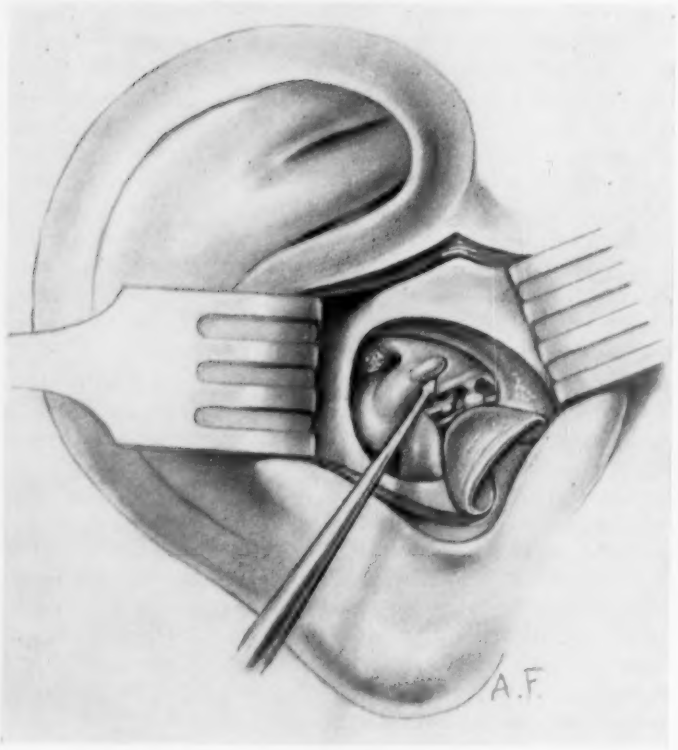


Fig. 3.—Cupola technique, Step 1, bony cupola being incised. The cupola technique for creating the fenestra nov-ovaiis renders the use of irrigation in the presence of the opened perilymph space unnecessary.

Through these observations and modifications of technique I was able over the years to eliminate the possibility that blood might become the cause of postoperative labyrinthitis. With these measures blood was prevented from entering the perilymph space and was also prohibited from starting within the perilymph space. In spite of this, however, I was unable to prevent in any of my patients the onset postoperatively of the clinical picture suggestive of serous labyrinthitis in various degrees of severity. It thus became obvious to me that, although it is true that bleeding into or within the perilymph space does cause postoperative labyrinthitis, it is also a fact that the most careful avoidance of blood entering into or starting within the perilymph space does not per se prevent postoperative onset of inflammatory reaction involving both the vestibular and the cochlear endolymphatic labyrinth, in varying degrees of severity. These observations led me to believe that blood within the perilymph space is not the only cause of postoperative labyrinthitis.

Still another readily recognizable technical factor which could be held responsible for the postoperative inflammatory reaction of the membranous endolymphatic labyrinth had to be considered. This was the trauma sustained by the delicate membranous walls of the endolymphatic labyrinth in our attempts to remove bone chips from the face of the membranous endolymphatic labyrinth with picks, after the use of the pulverization technique for creating the fenestra novovalis. Here again, however, change of operative method, using the cupola technique, whereby the bony cupola is removed intact without the formation of bone chips, thus avoiding the necessity of traumatizing the endolymphatic labyrinth, did not of itself serve to prevent the postoperative occurrence

Fig. 4.—Cupola technique, Step 2, bony cupola lifted and removed.

of inflammatory reaction in the endolymphatic labyrinth.

It became apparent then that neither the avoidance of bleeding into or within the perilymphatic space nor the avoidance of trauma of the endolymphatic labyrinth prevented the occurrence of postoperative inflammatory reaction of the endolymphatic labyrinth in any of the fenestrated patients. These two known factors which could be held responsible were possible to avoid. If, however, the constancy of the postoperative occurrence of serous labyrinthitis is to be explained, there must exist still another, hitherto-unrecognized technical error, constantly being made in the performance of the fenestration operation, which always causes various degrees of inflammatory reaction of the membranous labyrinth.

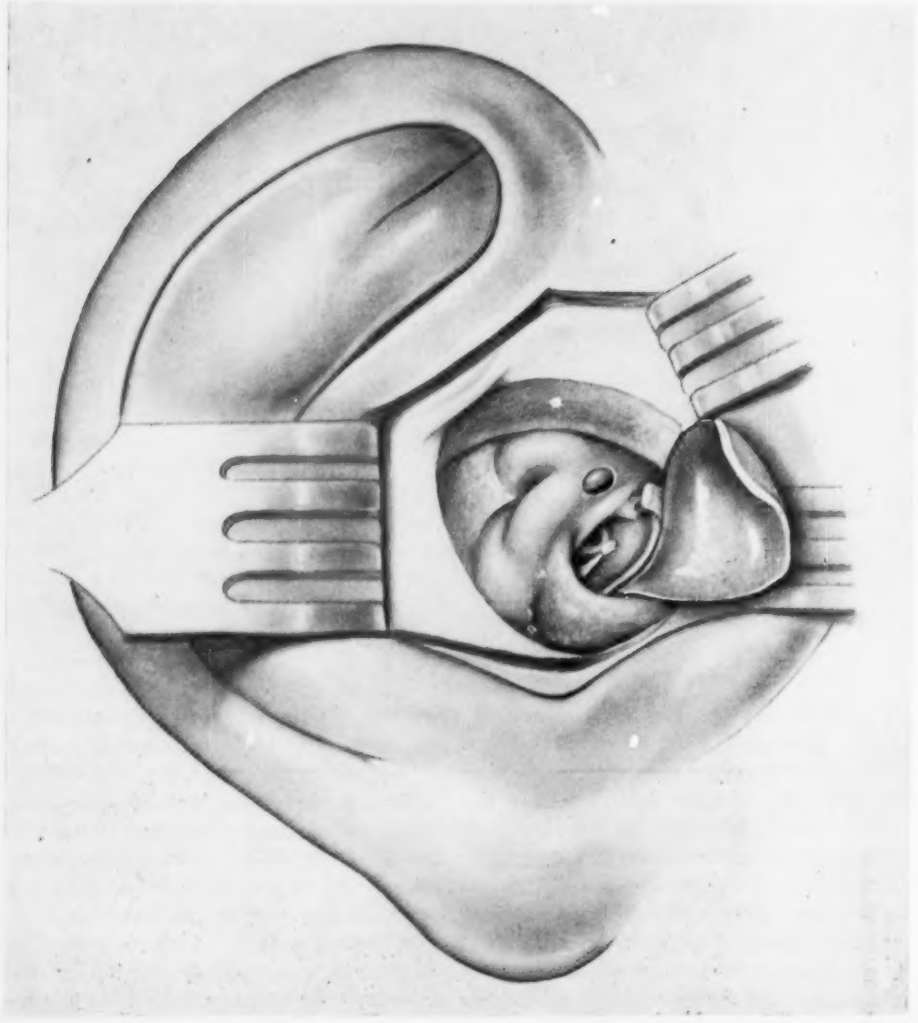
It occurred to me that irrigation and suction with any one of the physiological solutions used to date, for whatever purpose,

after having laid open the vestibular perilymph space, may be the one technical error always causing postoperative inflammation of the membranous endolymphatic labyrinth despite the careful avoidance of all the other technical errors which could cause it.

The postoperative inflammatory reaction may be either a chemical phenomenon produced by irrigation or a mechanical one produced by the suction accompanying the irrigation.

Irrigation in the region of the open perilymph space inevitably results in the replacement of the perilymph within the perilymph space with the fluid used for irrigation. The chemical difference of the fluid replacing the human perilymph may cause inflammatory reaction of the membranous endolymphatic labyrinth. Since the chemistry of the human normal perilymph is still unknown, there is no way of telling whether or not any physiological solution

Fig. 5.—Cupola technique, Step 3, open perilymph space, without the formation of bone dust or bone chips to be removed.



presently employed for irrigating the newly created fenestra region can effectively take the place of the natural perilymph which irrigation and suction removes from the perilymph space. It is not known if any of these solutions can be tolerated within the perilymph space for any length of time without causing inflammatory reaction of the delicate membranous endolymphatic labyrinth. None of the physiological solutions heretofore suggested for replacing isotonic saline solution or Ringer's solution for irrigating the perilymph space seemed to prevent the postoperative onset of inflammatory reaction of the endolymphatic labyrinth in varying degrees of severity.

The mechanical effect of the suction force upon the delicate membranous walls of the endolymphatic labyrinth cannot be dismissed without being suspected also as a possible cause of the postoperative inflammatory reaction of the membranous endolymphatic labyrinth.

Under the supposition that the effect of the chemical difference between the fluid used for irrigation and that of the human perilymph which has been replaced by it (within the perilymph space), as well as the mechanical effect of the suction force upon the membranous endolymphatic labyrinth, may be responsible for the postoperative inflammatory reaction of the endolymphatic labyrinth, I decided to conduct definitive tests. These tests would determine whether the constantly used irrigation and suction, with subsequent replacement of the perilymph with any of the currently used solutions which are different from perilymph chemically, can assuredly be held responsible for the constancy of postoperative serous labyrinthitis following fenestration surgery. In addition to avoiding the heretofore-proven causes of serous labyrinthitis I would also have to avoid irrigation and suction altogether after opening the perilymph space, in series of cases, in order to observe whether or not the postoperative labyrinthitis can thereby be consistently and consecutively prevented.

The tests were carried out on a series of 100 cases. I consecutively fenestrated 100 ears deafened as a result of otosclerosis, in the presence of good hearing by bone conduction, alternating between irrigation and nonirrigation, so that 50 cases were irrigated and 50 were not. The two previously proven causes of postoperative inflammation were carefully prevented. Bleeding into or within the perilymph space was prevented in every one of the 100 cases. By employing the cupola technique rather than the pulverization technique for creating the fenestra nov-ovalis there were no bone chips and no bone dust to remove from the perilymph space, and traumatization of the endolymphatic labyrinth was carefully avoided in all 100 cases.

The results appear conclusive. I observed that in the 50 cases where irrigation and suction was not used after opening the perilymph space there was no clinical evidence of postoperative inflammatory reaction of either the vestibular or the cochlear endolymphatic labyrinth. Of the 50 irrigated cases, however, all showed postoperative signs of labyrinthitis in various degrees of severity. I have thus proved to my satisfaction that irrigation and suction, after having laid open the vestibular perilymph space, is the constant cause of postoperative inflammatory reaction of endolymphatic labyrinth.

To place a still further and severer test on the role played by irrigation in postoperative labyrinthitis, I fenestrated three cases of fulminating otosclerosis in the presence of existing cochlear nerve function damage, as evidenced by the preoperative bone-conduction hearing loss. In all three of these I avoided irrigation and suction in the presence of the open perilymph space. To my surprise and great satisfaction there was no clinical evidence of postoperative inflammatory reaction of the endolymphatic labyrinth in any of these three cases, and in every one of them a hearing improvement to the level of the preoperative bone-conduction hearing was obtained (Figs. 6, 7, and 8). Formerly,

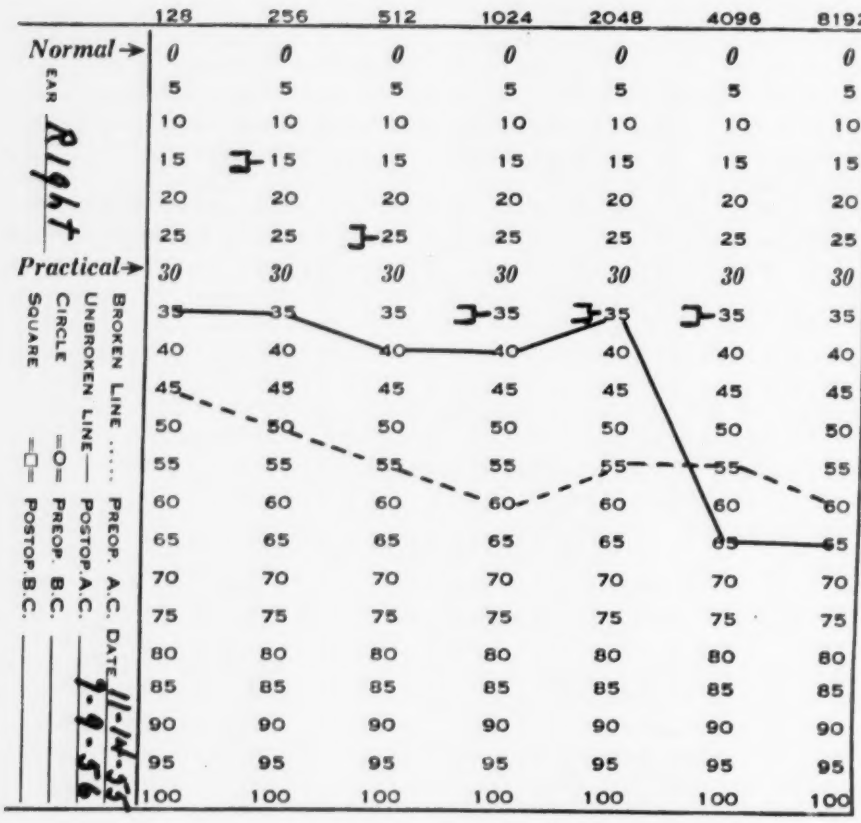


Figure 6

in cases of this type, the performance of the fenestration operation with the use of irrigation and suction after opening the perilymph space was followed by a prolonged postoperative period of severe vertigo and a marked depression of air-conduction and bone-conduction hearing which failed to improve, as a rule.

It thus became obvious that the fluid most physiologically suitable to surround the membranous labyrinth must be natural perilymph. It is apparent also that the best way to prevent postoperative inflammation of the endolymphatic labyrinth is to permit the labyrinth to continue bathing in Nature's perilymph instead of in any other solution which cannot possibly be made "physiological" to the membranous endolymphatic labyrinth so long as the chemical composition of the human perilymph remains unknown.

However, irrigation and suction after having opened the vestibular perilymph space can be avoided only by technically performing the fenestration operation without creating the need for irrigation once the perilymph space of the vestibular labyrinth has been laid open. The necessity for the use of irrigation after having opened the vestibular perilymph space can be avoided only by (1) using irrigation prior to opening the perilymph space for the removal of all bone dust and (2) employing the cupola technique instead of the pulverizing technique for opening the perilymph space.

Postoperative inflammatory reaction of the vestibular and the cochlear endolymphat-

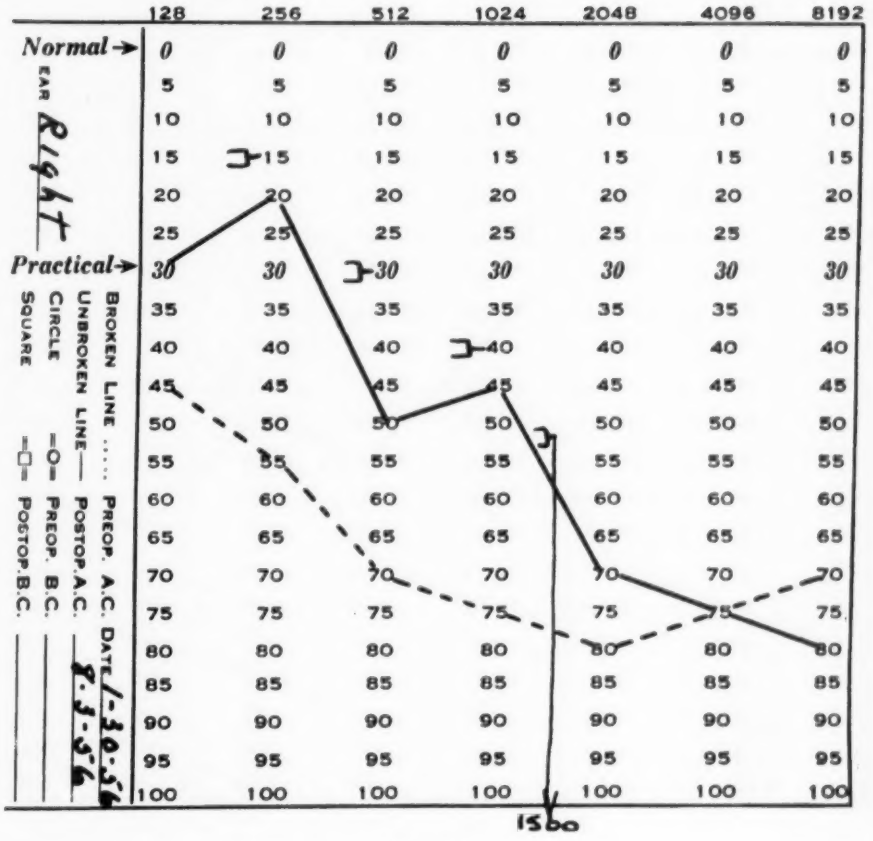


Figure 7

ic labyrinth can be continuously and consecutively prevented after the fenestration operation by employing the dry physiologically perfected fenestra nov-ovalis technique. This embraces the careful employment of the following technical precautions:

1. Use general and local analgesia instead of general anesthesia, to lessen bleeding.

2. Immediately control every individual bleeding point encountered.

3. Trim and tailor the tympanomeatal flap so that it will fall into place naturally and hug snugly each elevation and depression of the sculptured bony inner table of the surgically exenterated mastoid process, without leaving any air space for possible accumulation of serum underneath.

4. Remove all bone dust with irrigation before opening the perilymph space of the newly created fenestra.

5. Do not open the perilymph space until an absolutely blood-free and bone-dust-free surgical field has been obtained.

6. Avoid the necessity for removing bone chips and bone dust from within the perilymph space with picks, which could result in injury to and bleeding from the perilymphatic trabeculae. Use the cupola technique instead of the pulverization technique for creating the fenestra nov-ovalis.

7. Remove the bony cupola intact, in one piece, for opening the perilymph space.

8. Completely avoid irrigation after opening the perilymph space.

9. Cover the fenestra region and seal the perilymph space immediately after opening it with the thinnest, periosteum-lined portion of the tympanomeatal flap nearest to the tympanic portion thereof. Avoid using the periosteum-trimmed portion of the flap for sealing the perilymph space of the fenestra nov-ovalis.

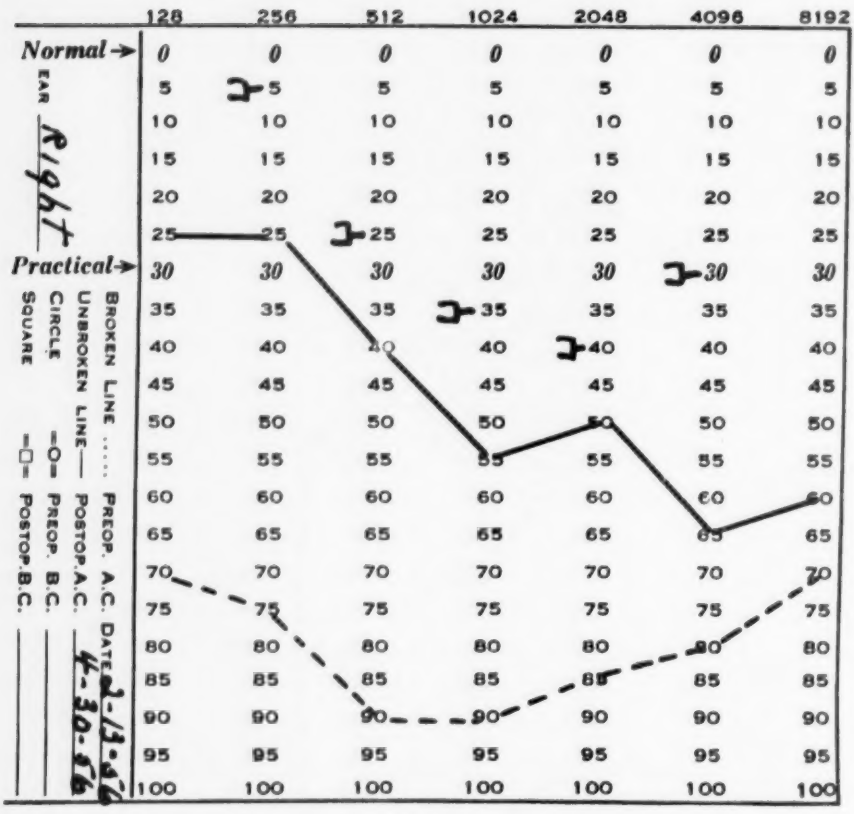


Figure 8

10. Invaginate the portion of the flap facing the fenestra into the fenestra gap sufficiently to contact and become adherent to the bony fenestra rim, in order to seal the perilymph space and prevent osteogenetic closure of the fenestra.

11. Pack mastoid wound carefully with paraffinated mesh gauze to hold the flap firmly in the position originally placed, in order to promote adhesion of the flap to the bony inner table of the surgically exenterated mastoid process.

Summary

The dry physiologically perfected fenestra nov-ovalis technique incorporates all the steps in the Lempert fenestra nov-ovalis technique, developed by me over the years, for permanently maintaining the improved hearing obtained with this operation. It also embraces new means for deliberately preventing serous labyrinthitis, the sequelae which heretofore, in many cases, was responsible for failure to realize the full measure of improved hearing to which suitable patients were entitled.

Postoperative inflammatory reaction of the vestibular and the cochlear membranous endolymphatic labyrinth following the fenestra nov-ovalis operation can be consistently avoided with the use of the dry physiologically perfected fenestra nov-ovalis technique. Since the prevention of postoperative osteogenetic closure of the fenestra nov-ovalis had been previously conquered and time-proven, the prevention of postoperative serous labyrinthitis, which has finally been conquered, results in removal of the final stumbling block to completely successful fenestration surgery.

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Visual and Other Factors Influencing Caloric Nystagmus in Normal Subjects

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Clinical experience has shown that the response to caloric stimulation may vary in the same person when the test is performed by different examiners, or even when the test is performed in the same manner by the same examiner on different days. The interindividual variations in response to caloric tests are even more striking.[1] This apparent inconsistency of response detracts greatly from the clinical usefulness of the caloric test. For this reason we set out to find factors that account for this variability of the caloric response, hoping that by a better understanding of these factors methods might be devised whereby caloric testing would produce more consistent results in practice.

It is immediately apparent that certain anatomic differences exist between persons that could account for their failure to respond to caloric stimulation in precisely the same manner. These include differences in the structure of the temporal bone and the thickness of the tympanic membrane. Such factors are difficult to assess and lie beyond the scope of this work.

The objective criterion of the response to caloric stimulation is the character of the nystagmus produced by it. The measurable factors of caloric nystagmus are ordinarily considered to be its (1) latency, (2) amplitude, (3) frequency, and (4) duration. It is most difficult to determine these factors accurately by simple direct observation, and we feel that the inadequacy of this method accounts for much of the variation in evaluation of caloric tests in practice. As a number of other investigators have also done, we have attempted to obviate this difficulty by graphically recording our results. Such graphic recording also permits us to determine two further factors of the nystagmic response, namely, (1) amplitude stability and (2) frequency stability, these being impossible to establish by means of direct observation alone.

The nystagmic response is a reflex mechanism dependent not only upon the integrity of the vestibular end-organs but also upon that of the vestibular nerve, vestibular nuclei, medial longitudinal fasciculus, and the third, fourth, and sixth cranial nerve nuclei and their nerves as well as the extraocular muscles themselves. As with other reflex mechanisms, we are not privileged to study the caloric response in isolation, since it is subject to integrative neural influences arising from the cerebrum, cerebellum, and upper part of the cervical cord. Among these neural influences we considered it likely that the state of visual fixation might play an important role in the nature of the nystagmic response obtained by caloric stimulation.

The state of visual fixation maintained by the patient during caloric testing varies with examiners. Some examiners decrease fixation by putting glasses with 20+ lenses on the patient during the test. Other examiners have the patient fix on a point 30 cm. distant, while still other examiners employ no fixation at all. We considered it possible that these differences in technique might

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account for some of the variability of response obtained from caloric tests. In addition to evaluating the role of visual fixation, we attempted to determine the influence of other cerebral factors (concentration and diversion) on caloric nystagmus.

Technique of Recording Caloric Nystagmus

The eye is a charged body, the cornea being electropositive with respect to the retina. Thus, when electrodes are placed on either side of the eye any movement of the eye in the plane of the electrodes will register a potential difference across them. The electrode approached by the cornea becomes positive with respect to the other.[2] This small potential (about 2 to 10μv. for each degree of movement) can be amplified and recorded by means of a penwriter, thus producing a permanent record of the activity of the eye in a given time. A number of workers[3,4] have utilized this technique successfully for recording eye movements.

Fig. 1.—Electrode arrangement employed in these studies. Solder disks are fastened about the eyes with collodion and filled with electrode jelly. With the eyes looking straight ahead, electrodes are located on a vertical line bisecting the pupil and equidistant from it. Electrodes are also placed at the inner and outer canthi of each eye. A standard interelectrode distance of 4 cm. for the vertically oriented electrode pairs and 6 cm. for the horizontally oriented electrode pairs was maintained.

Photographic and photoelectric techniques have also been devised.

We chose the electric method for recording eye movements in these experiments because it allowed relative freedom of movement for the subject and because it is the only method whereby eye movements can be recorded when the eyes are closed. Because of its availability, we employed a standard Grass electroencephalograph model 3B for our recordings. Our method most nearly approximates that used by Bergman and associates.[3] Solder disks were fastened about the eyes by means of collodion and filled with electrode paste, in the same manner that electrodes are applied in electroencephalography. Electrodes were placed at the outer and inner canthi of each eye and above and below the eyes on a vertical line bisecting the pupil, with the eyes looking straight ahead. A standard interelectrode distance of 6 cm. for the horizontal lead and 4 cm. for the vertical lead was maintained. This was found to be a fairly close eye lead for most subjects. The left and right vertical electrode pairs were recorded on channels 1 and 2 of the electroencephalograph, and the left and right horizontal pairs were recorded on channels 3 and 4. Figure 1 illustrates the electrode arrangement.

We were thus able to record not only the relative amplitude of the excursion of the eyes but also the direction in which they moved, since each direction of movement produced waves of a different phase relationship.

Since the potential produced by a given number of degrees of rotation of the eyes varies greatly from one subject to another, it was necessary to determine this potential for eye movements of a known number of degrees in each subject individually. This was accomplished by having the subject look from one point of light to another, these lights being fixed on a board at known distances apart, and recording the waves so produced by means of the electroencephalograph. The angle of rotation

of the eyes thus became a tangent function of the distance of the light board from the subject and the distance between the two points of light on the board. From these data we were able to determine the potential produced by an ocular rotation of known amplitude and could derive the potential in microvolts produced by each degree of ocular movement.

It should be noted that the amplifiers used in electroencephalographic equipment are capacitance-coupled and therefore do not record steady potentials. When a steady potential is applied across the inputs of the EEG, the pen is deflected to record the potential difference, then falls slowly back to the base line at a rate dependent upon the time constant of the amplifier system. For this reason our records show movement of the eyes only, and not the position in which the eyes are held. The velocity of eye movement is such, however, as to fall well within the time constant of EEG equipment, so that there is no significant distortion of the wave forms produced by nystagmus as a result of this factor.

Method

Upon completion of the measurements of the eye movements in degrees and microvolts, the subject was seated in a reclining chair and the head tilted backward so that a line from the external ear canal to the outer canthus of the eye was in a vertically inclined plane of 60 degrees, as described by Hofer to be the optimal position for eliciting caloric nystagmus from stimulating the horizontal semicircular canal. The subject's head was maintained in a constant position, equidistant from the shoulders, throughout the procedures.

Five caloric tests were performed on the left ear of each subject using a modification* of Kobrak's[6] minimal caloric test introducing 5 cc. of ice water at 40 F into the ear. The water was injected into the left external auditory canal over a period of 10 seconds, at least 5 minutes being allowed to elapse between tests. This interval seems adequate, since Lorente de Nó[6] and others[7] carried out continuous labyrinthine caloric stimulation in animals over a period of hours on successive days without observing any diminution of response.

All subjects used were persons between the ages of 21 and 35 years, in good general physical condition, and free of any past history of disorders of the ears, eyes, or vestibular systems. The subjects were examined prior to tests for evidence of spontaneous nystagmus and abnormalities of the external auditory canal or tympanic membrane. Eighteen subjects were studied.

Caloric tests were performed under the following conditions: (1) with fixation at 30 cm. (the subject was asked to fix on the end of a pencil held 30 cm. from the eyes); (2) gazing at infinity (the subject looked down an empty corridor at a blank wall more than 50 ft. away); (3) wearing Frenzel[8] glasses (the subject wore a pair of goggles with 15+ lenses illuminated from the back so that nothing was visible through them); (4) with eyes open, but in dark (Ace bandages applied over the Frenzel glasses so as to exclude all light), and (5) with eyes closed (the subject was asked to close the eyes lightly, as in sleep).

At intervals during the reaction following caloric stimulation, the subjects were occasionally asked to close their eyes for brief periods and were given simple arithmetic and spelling problems, or were alerted by stimuli such as unexpected sounds, a pinch, or a blast of compressed air on an exposed part of the body.

Results

The Table gives the average amplitude, frequency, and duration of caloric nystagmus under the conditions of fixation previously mentioned among 18 subjects.

The duration of nystagmus was measured from the first detectable nystagmic deflection to the last observable nystagmic wave. In nearly all instances the beginning and the end-point of the nystagmic response were readily apparent. It is interesting to note that often small electrically recorded

Average Amplitude, Frequency, and Duration of Caloric Nystagmus Recorded in Protocol

	Amplitude (degrees)	Frequency (seconds)	Duration (seconds)
Fixation at 30 cm. (18 subjects)	3.3	3.1	102
Eyes gazing into infinity (14 subjects)	6.0	2.3	119
Frenzel glasses (16 subjects)	12.0	2.2	132
Eyes open in dark (16 subjects)	13.0	2.2	155
Eyes closed (18 subjects)	19.0	1.9	184

* Mayo Clinic modification of Kobrak's minimal caloric test.

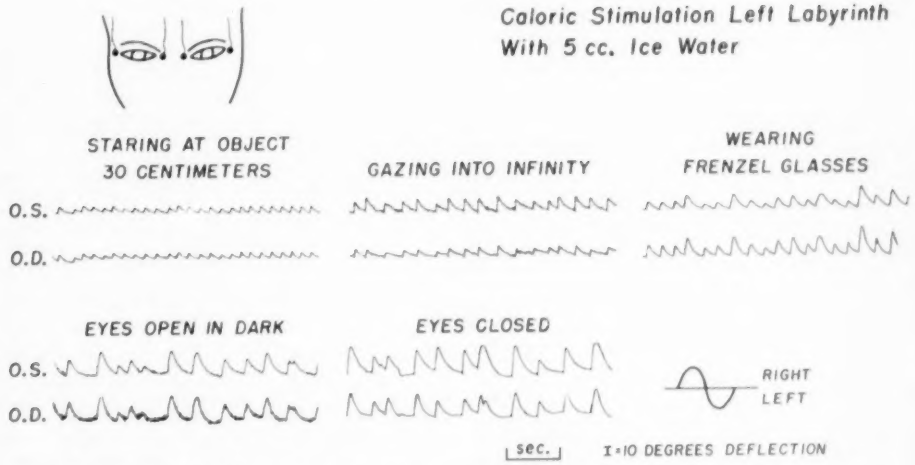

Fig. 2.—Variations in amplitude and frequency of caloric nystagmus with changing influence of visual fixation in a normal subject. Note the increase in amplitude and decrease in rate of caloric nystagmus as visual influence is removed. The patterns illustrated are quite constant, occurring continuously in the record so long as the conditions of fixation are maintained.

nystagmic waves appeared on the record before the nystagmus was clinically detectable.

The amplitude and frequency of the caloric nystagmus are expressed as a mean average of the amplitude and frequency of the nystagmic excursions during the period of maximal amplitude response. This period was measured from the first point of maximal response to the first detectable decrease in maximal amplitude.

As illustrated in the Table, there is an inverse relationship between the amplitude and the frequency of the nystagmus. The amplitude increases and the frequency decreases progressively with increasing interference with fixation. There is also a moderate, parallel increase in the duration of nystagmus with decrease in the fixation factor.

Figure 2 illustrates the characteristics of nystagmus under different conditions of fixation as they appeared in the records. During fixation the nystagmus is fine, rapid, and regular. It has good frequency and amplitude stability. The waves produced are of saw-tooth contour, having definite quick and slow components.

With the subject's gaze at infinity the nystagmus remains rather fine and rapid, but there is some loss of amplitude and frequency stability.

When the subject is wearing Frenzel glasses or has the eyes open in the dark, the type of nystagmus seen is roughly comparable under these two conditions. The frequency is further decreased and the amplitude greatly increased. There is poor amplitude and frequency stability. There is a slight increase in the duration of the quick component.

With eye closure, further slowing of frequency and increase in amplitude of nystagmus occur. Frequency and amplitude stability is very poor. In 10 of the 18 subjects nystagmus during eye closure was intermittent, occurring in brief bursts, lasting 4 to 10 seconds, between which were short periods of inhibition.

In a few instances nystagmus failed to appear during eye closure until activated by alerting the subject in some way, and well-developed nystagmus during eye opening was often completely inhibited by eye closure.

Figures 3 and 4 illustrate some of the effects of eye closure, mental activity, and alerting stimuli on caloric nystagmus.

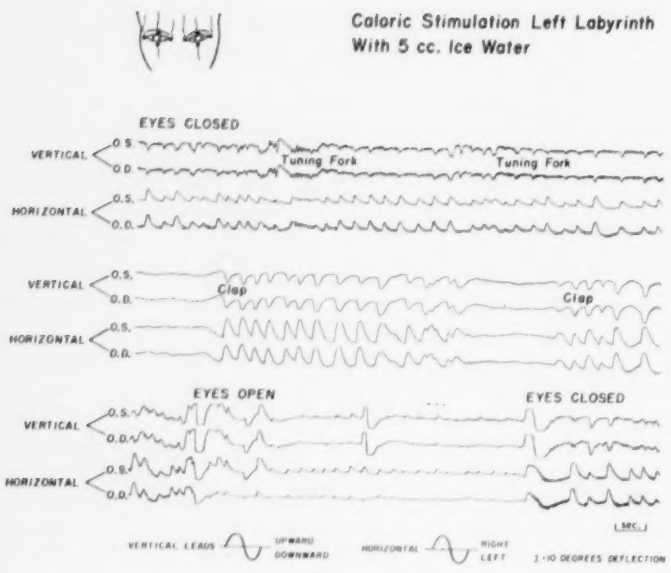

Fig. 3.—Effect of eye closure and alerting on caloric nystagmus. In the upper recording, a tuning fork applied over the ear produces a startle or alerting reaction, depressing the caloric nystagmus when the eyes are closed. In the middle recording, nystagmus, which is absent with the eyes closed, appears for a brief burst following an alerting reaction produced by clapping the hands. In the lower recording is shown the decrease in amplitude and increase in rate of caloric nystagmus with the eyes open compared with nystagmus with the eyes closed.

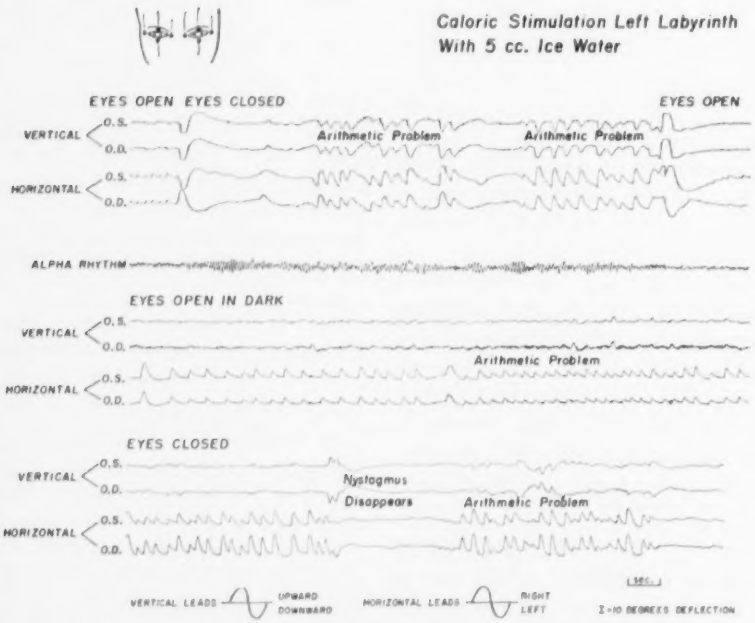

Fig. 4.—Effect of eye fixation and calculation on caloric nystagmus. The upper recording shows how nystagmus disappears with eye closure and reappears with calculation for a brief burst, disappears, and again reappears with further calculation; changes in the alpha rhythm with the eyes open and closed are recorded below. The middle recording shows the change that occurs with calculation when the eyes open in the dark. In the lower recording note how nystagmus disappears with the eyes closed and reappears for a brief burst with calculation and again disappears.

Comment

Three known central nervous mechanisms are involved in the voluntary and reflex control of eye movements. They are (1) the voluntary motor system; (2) the occipital reflex, or following, system, and (3) the vestibulocerebellar, or proprioceptive, system. These are paired systems, left and right, and each functions in such a way that its influence can be thought of as pushing the eyes toward the opposite side. Voluntary eye movements are under control of cells located in the posterior portion of the second frontal convolution.[9] The suprasegmental pathway passes through the anterior limb of the internal capsule near the genu and continues with other corticobulbar fibers into the pons, where some of the fibers pass with the medial lemniscus of the same side.[9] The fibers cross over to the third and sixth cranial nerve nuclei of the opposite side near their respective levels. Whether or not fibers to the fourth nucleus cross is unknown. Unilateral cortical and subcortical lesions involving this pathway produce a temporary conjugate deviation of the eyes to the side of the lesion.[10]

The more primitive suprasegmental reflex mechanisms controlling the eyes are a function of the occipital cortex.[10] These include following movements, fixation and refixation, and the visual blink reflex. Electric stimulation of the peristriate area produces conjugate deviation of the eyes to the opposite side. The fixation-refixation pathway is probably by way of the optic nerve and tract to the lateral geniculate body and from there to the calcarine cortex, then to the peristriate area, from which fibers pass to the third and sixth cranial nerves of the eye on the opposite side by way of the occipitomesencephalic (internal corticotectal) tract. Lesions involving these pathways may cause the eye to oscillate about the fixation point and to wander aimlessly, as is seen in some totally blind eyes.[10]

The labyrinths exert an influence on eye movements by way of the vestibular nuclei and the medial longitudinal fasciculi to the third, fourth, and sixth cranial nerve nuclei. There is also an indirect influence from the flocculonodular lobe and the anterior lobe of the cerebellum. The cerebellum and basal ganglia exert influences on the extraocular muscles much the same way as they do on other muscles of the body. If one labyrinth is destroyed, a slow tonic movement of the eyes to the same side is temporarily produced owing to the unopposed action of the other labyrinth. If the cerebral hemisphere of the side of the lesion is intact, a quick compensatory movement in the opposite direction occurs and thus a nystagmus is produced.

It is thought that nystagmus, like its subjective counterpart, vertigo, results when the above-mentioned mechanisms receive and transmit conflicting information which cannot be fully integrated by the nervous system. Thus, when the balance of labyrinthine control is altered by injection of cold water into one ear, a slow tonic movement of the eyes to the same side is produced. The opposite cerebral hemisphere, acting to maintain the eyes in the primary position of gaze, causes a quick movement of the eye in the opposite direction and thus a nystagmus is produced. This cerebral component of labyrinthine nystagmus has been considered a function of the voluntary motor system. Our evidence, however, suggests that it results from a compounding of influences involving the voluntary frontal mechanism, reflex following (the occipital mechanism), and perhaps other cerebral influences not yet well understood which have to do with orientation of the eyes. Thus, when fixation is fully developed during caloric testing, we get a nystagmus of the highest frequency and the greatest degree of amplitude and frequency stability. As fixation and vision are progressively reduced, as when the subject wears the Frenzel glasses or has the eyes open in darkness, we get a nystagmus of slow frequency, and less frequency and amplitude stability. With eye closure additional cerebral influences seem to come into action, for

under this circumstance caloric nystagmus is further slowed in frequency or is even abolished. It would appear that with eye closure the eyes may no longer remain in the primary position because the extraocular motor nuclei are then under the influence of stimuli from brain-stem nuclei, chiefly vestibular, and are uncontrolled by cortical-inhibiting influences. With alerting or mental activity, even when the eyes are closed, it would appear that this tendency of the eyes to maintain themselves in the primary position returns, since we have been able to demonstrate fairly consistently the appearance of bursts of nystagmus during periods of alerting or problem solving.

An alternate explanation would be that eye-centering mechanisms of the cerebral cortex hold in check caloric nystagmus during eye closure only so long as the attention of the person can be entirely directed toward this end. Any diverting influence, such as mental activity or sudden unexpected stimuli, interferes with this function of the cortex.

The alteration of the status of the central nervous system that takes place upon eye closure is not well understood. That change does occur is certain, however, as evidenced by the frequency with which alpha activity appears in the electroencephalogram with eye closure. It is interesting that caloric nystagmus should be facilitated by two important factors that rather consistently inhibit the alpha rhythm, that is, eye opening and alerting. The consistency with which mental activity and alerting stimuli were observed to facilitate caloric nystagmus during eye closure appears to bear out and still further establish the assumption made by Mowrer [11] and by Fischer and Wolfson [12] that tension factors might influence the caloric response. Spiegel and Aronson [7] had previously observed the appearance of nystagmoid movements in successive bursts or groups intermixed with complete absence of nystagmus following caloric stimulation during eye closure. They suggested that these fluctuations may be due to fluctuations in the excitability of the reflex arc itself. Our findings corroborate theirs and would seem to support this thesis.

The foregoing observations have demonstrated that electrographic recording allows for considerable increase in the sensitivity and accuracy of caloric testing as compared with presently employed visual methods. The application of such a technique in further research studies of diseases of the labyrinth, vestibular nerve, and vestibulocerebellar pathways might yield information useful to the clinician in the diagnosis of these diseases. For this purpose a simplified electrode array that could be rapidly applied for graphic recordings of caloric nystagmus might ultimately increase the accuracy and extend the usefulness of the caloric test in practice.

Summary

The caloric responses of 18 normal subjects were recorded electrographically. Observations were made regarding the effect of (1) fixation, (2) vision, (3) eye closure, and (4) alerting and mental activity on caloric nystagmus. As the influence of fixation was removed, there was a decrease of the rate and an increase of the amplitude of caloric nystagmus. Parallel with this change a decrease in frequency and amplitude stability occurred. The average duration of the nystagmus was also moderately increased with decrease in the fixation factor.

Eye closure increases the foregoing changes. Under this condition caloric nystagmus is often completely inhibited or is present only intermittently, appearing in brief bursts.

Mental activity or alerting frequently elicited a brief burst of nystagmus when the nystagmus had been inhibited by eye closure. It is possible that alerting increases the tendency of the eyes to orient themselves in the primary position during eye closure or that alerting interferes with cerebral inhibition of nystagmus. We have pointed out the inverse relationship of these bursts of

nystagmus to the alpha rhythm in the electroencephalogram. This finding suggests that cerebral factors play an important role in caloric nystagmus and supports the suggestion of others that the caloric response may vary with the patient's state of alertness.

Mayo Clinic

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A Study of One Hundred Cases of Neck Dissection

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The term "prophylactic" neck dissection is used to define an operation performed in the absence of clinically demonstrable cervical metastases. This is in contradistinction to a "curative" (radical) neck dissection in which there is demonstrable lymphadenopathy, presumably metastatic.

The use of "prophylactic" has been objected to by some,[1] and they propose the term "dissection for probable small metastases." However, by common usage "prophylactic" is generally accepted.

There is almost universal agreement that a radical neck dissection is indicated for the patient with a curable primary malignancy of the head and neck with cervical metastases. However, the opinion is divided as to the advisability of a neck dissection in the absence of demonstrable lymphadenopathy. There are three main schools of thought relative to this subject. The first advocates routine prophylactic neck dissection, the second believes that such a procedure is never indicated, while the third prefers prophylactic neck dissection in selected cases.

Those who recommend a routine prophylactic neck dissection[1-5,22] believe that intraoral cancer should have the same management as cancer in other areas, such as the breast and rectum. They state that the cure rate in these latter locations increased when it became the routine policy to excise the regional nodes whether clinically enlarged or not. They also condemn the "wait-and-see" method because of the rapidity with which neck metastases sometimes develop, the reluctance of some patients to submit themselves to another operation after the primary lesion has been controlled, and because of many patients' failure to keep in contact with the physician for repeated observation.

The group which opposes any prophylactic neck dissection[6-14] believes that its routine use is illogical and unacceptable because it leads to a great deal of unnecessary surgery. They claim that 15 out of 16 such operations are useless in cases of carcinoma of the lip and 4 out of 5 are useless in cases of carcinoma of the tongue. They also believe that a policy of routine prophylactic neck dissection does not affect to any appreciable degree the over-all cure rates. Furthermore, they think that a prophylactic operation gives a false sense of security to the surgeon. One author[17] found a 10% lymph node recurrence after 247 prophylactic neck dissections. This group has carefully studied the problem in relation to cancer of the lip and tongue and believes that such a rationale also applies in other forms of oral, pharyngeal, and laryngeal carcinoma.

The third group, to which we belong, believes as stated in a previous communication[16] that the young surgeon cannot be given constant rules in regard to protective neck dissections. We can only retire with little grace behind those two old excuses "experience" and "judgment." Two theoretical cases may help to clarify our opinions in relation to this problem. We would not do a prophylactic neck dissection in the case of an 80-year-old man presenting a fungating, 5 mm., slowly enlarging lesion of his lower lip without palpable nodes if the primary neoplasm proved to be well differentiated and noninvasive on microscopic examination and if the patient could be

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examined for metastases at frequent intervals. However, if a 30-year-old man presented himself with a rapidly growing primary lesion of similar size, we would strongly recommend bilateral prophylactic neck dissection, especially if the carcinoma proved invasive and not well differentiated on microscopic examination. We would not advise merely a suprahyoid procedure but would advocate a bilateral supraomohyoid dissection. When intraoral lesions are encountered, especially of the tongue or floor of the mouth, our leanings toward prophylactic neck dissection become more pronounced.

Among laryngologists a great deal has been written in relation to the advisability of a prophylactic neck dissection in carcinoma of the larynx.[18-21] It has been reported that neck metastases appeared in 30% of the patients who had laryngectomy without clinical evidence of cervical adenopathy at the time of the examination or operation.[18-21] Because of this, one author [18] has advised a prophylactic neck dissection in every laryngectomized patient. However, the general consensus, with which we agree,[19-21] is that the prophylactic neck dissection should be reserved for patients whose laryngeal carcinoma is located in the aryepiglottic fold, ventricle, epiglottis, and subglottic region. In some cases we recommend a prophylactic unilateral or bilateral neck dissection, depending upon the location of the lesion. We feel that a prophylactic neck dissection need not be done in patients in whom the carcinoma is entirely confined to the true vocal cord.

TABLE 1.—*Location of Primary Lesion*

Group I	No.	%
Larynx	11	22
Gingiva	10	20
Tongue	7	14
Floor of mouth	5	10
Tonsil	5	10
Buccal mucosa	3	6
Face	3	6
Nasopharynx	2	4
Soft palate	2	4
Lower lip	1	2
Undeterminate	1	2
Total	50	100

Group II	No.	%
Lip	32	64
Esophagus	3	6
Thyroid	3	6
Face	3	6
Parotid	2	4
Buccal mucosa	2	4
Submaxillary gland	1	2
Eye	1	2
Branchiogenic	1	2
Primary of neck	1	2
Primary unknown	1	2
Total	50	100

TABLE 2.—*Sex*

Group I	No.	%
Male	45	90
Female	5	10

Group II	No.	%
Male	45	90
Female	5	10

In the previously mentioned article [16] we expressed our clinical impressions. However, no specific clinical data were given in that communication, so we studied 100 cases of neck dissections in order to reexamine our opinion. Of these 100 patients, 50 had a radical neck dissection performed either by one of us or under our supervision. The other 50 patients were operated upon in our institution by another department where routine prophylactic neck dissection is favored. Thirty-five of these patients had a bilateral supraomohyoid neck dissection.

The group composed of 50 cases of therapeutic radical neck dissections was designated Group I, and the group in which the majority of the dissections were done as prophylaxis was designated Group II. When comparing these two groups the following observations were made: In relation to the primary site of the lesion it was found in Group I that the larynx, gingiva, tongue, and floor of the mouth accounted for 66% of the cases, while in Group II the primary lesion was located in the lip in 64% of the cases (Table 1).

In relation to sex distribution, both groups presented identical figures; 90% of the cases were seen among males and 10% among females (Table 2).

TABLE 3.—*Age Incidence*

Group I	No.	%
3rd decade	1	2
4th decade	10	20
5th decade	6	12
6th decade	20	40
7th decade	12	24
8th decade	1	2

Group II	No.	%
1st decade	1	2
2nd decade	1	2
3rd decade	1	2
4th decade	1	2
5th decade	6	12
6th decade	19	38
7th decade	17	34
8th decade	4	8

TABLE 4.—*Pathology of the Primary Lesion*

Group I	No.	%
Epidermoid carcinoma	(48)	(96)
Grade 1	2	4
Grade 2	33	66
Grade 3	9	18
Grade 4	4	8
Adenocarcinoma	1	

Group II	No.	%
Epidermoid carcinoma	(38)	(76)
Grade 1	5	10
Grade 2	23	46
Grade 3	9	18
Grade 4	1	2
Intermediate ca.	2	4
Adenocarcinoma	6	12
Melanoma	2	4
Fibrosarcoma	1	2

The study of the age at time of operation disclosed that in Group I 64% of the patients were in their sixth and seventh decades, while these age groups accounted for 72% of the cases in Group II (Table 3).

A review of the pathology of the primary lesion showed that epidermoid carcinoma accounted for 96% of the cases in Group I and 76% in Group II (Table 4).

The investigation was also directed toward the pathology of the metastases, and it was found that 94% of the cases in Group I were epidermoid carcinoma, while only 46% of the cases presented this type of lesion in Group II (Table 5).

The incidence of microscopically positive nodes was 96% in Group I, while only 66% of Group II had microscopically positive nodes (Table 6).

TABLE 5.—*Pathology of Cervical Metastases*

Group I	No.	%
Epidermoid carcinoma	(47)	(94)
Grade 1	0	0
Grade 2	31	62
Grade 3	10	20
Grade 4	6	12
Adenocarcinoma	1	2

Group II	No.	%
Epidermoid carcinoma	(23)	(46)
Grade 1	2	4
Grade 2	15	30
Grade 3	5	10
Grade 4	1	2
Intermediate ca.	1	2
Adenocarcinoma	6	12
Melanoma	2	4
Fibrosarcoma	1	1

The five-year survival rate in Group I was 30%, while in Group II it was 31% (Table 7).

An evaluation of the results of our neck dissections, in spite of the small number of cases studied, signifies to us that cure rates are not appreciably increased by use of rigidly routine prophylactic procedures. Reservation of the operation for selected cases eliminates a large number of unnecessary operations and the occasional sequelae and complications that are inherent to any surgical intervention.

Summary

The rationale of the three main schools of thought relative to the problem of prophylactic neck dissection is presented.

The results in 100 cases of neck dissection are analyzed.

In our opinion, prophylactic neck dissection should be advised in specific cases but neither advocated as a routine procedure nor condemned as a useless operation.

TABLE 6.—*Incidence of Microscopically Positive Cervical Nodes*

Group I	No.	%
Incidence of microscopically positive nodes	48	96
Incidence of microscopically negative nodes	2	4
Group II		
Incidence of microscopically positive nodes	33	66
Incidence of microscopically negative nodes, of which 15 were cases of carcinoma of the lip	17	34

TABLE 7.—*Five-Year End-Results*

Group I	No.	%
	Indeterminate cases	
During 5-yr. follow-up, patients died of causes not related to carcinoma, primary was not controlled, or nodes were negative	10	20
	Determinate cases	
Total No. of cases (40)		
Alive	12	30
Dead	28	70

Group II	No.	%
	Indeterminate cases	
During 5-yr. follow-up, patients died of causes not related to carcinoma, primary was not controlled, or nodes were negative (this includes 17 cases with negative nodes)	21	42
	Determinate cases	
Total No. of cases (29)		
Alive and well	9	31
Dead	20	69

A routine policy of prophylactic neck dissection did not increase the five-year survival rate in our series.

A great deal of unnecessary surgery is performed whenever a rigid routine or prophylactic neck dissection is followed.

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Facial Rehabilitation Following Radical Parotid-Gland Surgery

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Two great deterrents have hindered the successful management of tumors of the parotid gland. The first has been a lack of accurately coordinating knowledge concerning the microscopic classification of parotid tumors, their biology, management, and prognosis. Recent publications have helped to clarify this deficiency.[1] The second deterrent has been fear of injury to the facial nerve and the use of inadequate methods of rehabilitation of the paralyzed face. A more vigorous approach to this latter problem has proved advantageous to the management of these tumors and helpful to the patient.[2]

The vast majority of tumors of the parotid gland can be cured without permanent injury to the facial nerve. This includes all the benign tumors and cysts, which comprise approximately 70% of the tumefactions in this gland. Of the remaining one-third, which are malignant tumors, the nerve need not be permanently damaged in 10%-15% of the cases. This, then, leaves 15%-20% of the patients where the facial nerve may be totally or partially resected and rehabilitation of some degree is indicated.

Only rarely is the facial nerve cut in the extirpation of benign tumors, of which approximately 60% are benign mixed tumors. This single fact establishes the basis and technique of treatment. If the benign tumor (or cyst) is small and limited to the lateral lobe, a partial lateral lobectomy is done, ordinarily without fear of injury to the nerve, and usually without the aid of faradic testing. If the benign tumor (or cyst) is large and occupies the major portion of the lateral lobe, a total lateral lobectomy is done along with identification and faradic testing of the facial nerve. If the benign tumor (or cyst) originates in the isthmus or medial lobe, a total parotidectomy is performed with identification and testing of the facial nerve, and careful dissection. In this latter instance, there is a temporary weakness of the facial muscles for several weeks to several months, due to trauma, stretching, and dissection along the nerve. When only a lateral or partial lobectomy is performed, there is usually no facial-nerve weakness. Occasionally a benign tumor will become so large and so enmesh the nerve in its growth that total removal without temporary paresis is impossible. Under these circumstances the tumor is cut to free the nerve filaments as the dissection progresses.

The use of faradic stimulation as a method of identification and testing of the facial nerve has recognizable limitations. Although this testing has considerable value, in that the vast majority of nerves do respond to its direct application, thus affording valuable information, some nerves do not respond. In this latter circumstance great care must be exercised not to accept the lack of response as evidence that the structure tested is not a nerve and that it is safe to cut it. If a structure resembles a nerve, it should not be comprised on the basis of a negative test, but should be preserved until further evidence reveals itself

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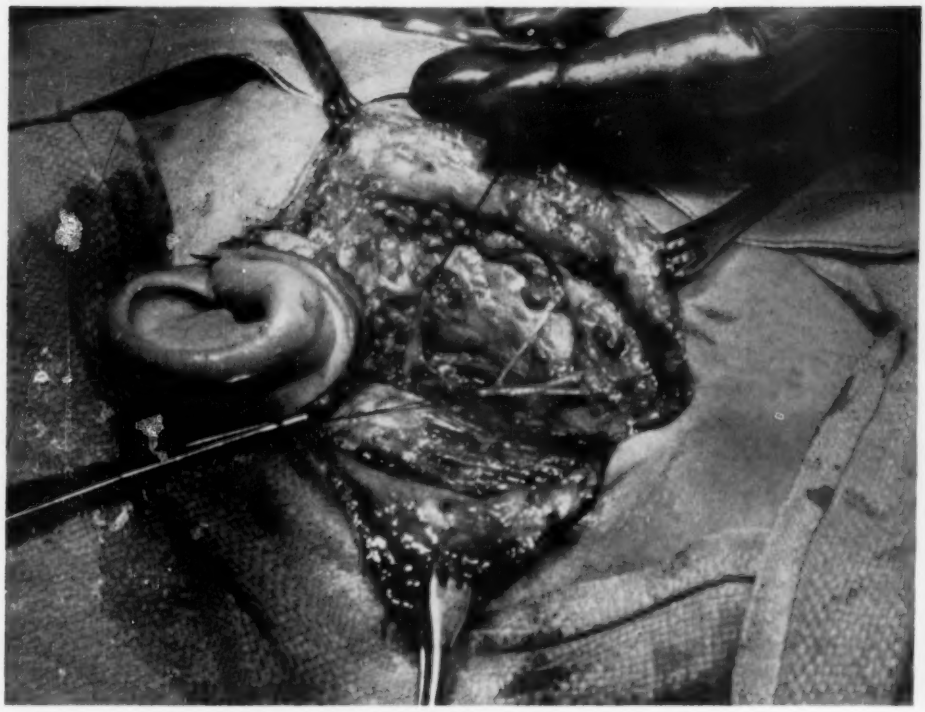


Fig. 1.—Resection of parotid gland and preservation of facial nerve.

in the dissection. Two factors seem to explain this phenomenon. Certain anatomical "blind spots" exist in normal nerves, and these areas do not respond to minimal faradic stimulation. In nerves that have been stretched or traumatized, there is the element of edema and nerve fatigue in preventing a response.

Not all malignant tumors of the parotid gland require resection of the facial nerve. The acinous-cell adenocarcinoma and low-grade mucoepidermoid carcinoma are totally resected with the entire parotid gland with salvage of the nerve trunk intact, unless it is specifically invaded by the tumor. This procedure is deemed warranted in view of the low mortality rates of these tumors (15% and 10%, respectively). Small, highly malignant tumors of the adenocarcinoma, squamous-cell carcinoma, mucoepidermoid carcinoma, and malignant mixed tumor types, limited to the lateral lobe and not involving the facial nerve, are treated by block dissection of the neck and total parotidectomy, without permanent injury to the nerve. If these highly malignant tumors occupy the major portion of the lateral lobe or if they originate in the middle lobe or isthmus, it is usually impossible and unwise, because of their high mortality rate (60%-80%), to attempt to salvage the facial nerve. These patients demand active facial rehabilitation.

A classification of the highly malignant tumors is as follows:

1. Malignant mixed tumor. This tumor is found in 5%-10% of the benign mixed tumors, occurring in a slightly older age group, more frequently in benign tumors that have been operated upon before, and frequently appearing in benign tumors that have been quiescent for years. The prognosis for cure of this tumor is poor, having approximately a 50% mortality. It has great facility for metastasizing to the neck,

throughout the body, and for local recurrence.

2. Mucoepidermoid carcinoma, high-grade type. Clinically, this tumor is more aggressive, growing rapidly, ulcerating the skin, causing local pain, and eventually invading the facial nerve. It has a mortality of approximately 70%, with a high incidence of metastasis to the neck, general spread, and local recurrence.

3. Adenocarcinoma. This group of tumors is composed of the adenocystic, mucous-cell, anaplastic, solid or trabecular, and pseudoadamantine types of carcinoma. They are all clinically aggressive, growing rapidly, and causing pain, ulceration of the skin, and eventual facial-nerve paralysis. The mortality is in the neighborhood of 70%, with a great tendency to metastasize to the neck, throughout the body, and to recur locally.

4. Squamous-cell cancer. This type of cancer is very aggressive clinically, with rapid growth, ulceration of the skin, local pain, and eventual seventh-nerve paralysis. It has a mortality of approximately 80%, with a great tendency to recur locally and to metastasize to the neck but, curiously, not manifesting extensive generalized spread.

Facial Rehabilitation

The technique of parotid-gland resection will not be discussed in this paper, as it has been covered in a previous report.[2] When it has been necessary, however, to section or resect the facial nerve, five technical procedures are available to assist in the rehabilitation of the paralyzed face.

1. Facial suspension. This is perhaps the least satisfactory of all the methods of facial rehabilitation, since the face appears normal, in an inert way, only in the position of repose. The affected side of the face is in a state of complete paralysis and is completely devoid of intrinsic muscular activity. This method should be used only as an aid to other techniques, and as a last resort when other methods are unwarranted or have failed. The paralyzed face may be suspended by the implantation of autogenous or homologous fascia lata about the lips, oral commissure, and orbit, and securing the strips into the temporal fascia and muscle. When this masticatory muscle is contracted, extrinsic movement of the face occurs. This movement is minimal and not in essential harmony with facial expression.

Autogenous fascia lata is preferred to any other type. Overcorrection in the suspension is essential to accommodate subsequent relaxation. Fascia lata may also be used to suspend the face by threading it through drill holes in key positions of the zygoma and fixing it to the lips and oral commissure. Inert metallic wires may be used for the same purpose of suspension but have a greater tendency to eventually permit relaxation. Movement is completely absent in this latter technique.

2. Muscle rotation. This technique has value as an individual procedure and as an adjunct in nerve grafting. The masseter and temporal muscles are available. The transposition of the temporal muscle and coronoid process of the mandible just above the oral commissure gives good tone to that side of the face and active physiological suspension. The transposition of the anterior half of the masseter muscle into the region of the oral commissure and lips supplies active physiological suspension of the face and moderate movement upon mastication. Either one or both of these muscles may be used for rotation purposes when complete facial-nerve grafting is anticipated. This technique supplies the face with animation during the year in which the facial nerve is regenerating, and is a routine part of the technique. Muscle rotation is not indicated in simple nerve resuturing.

3. Facial balance. When the restraining forces of expression are eliminated on one side of the face by a permanent partial or complete paralysis of the facial nerve, the contralateral muscles contract excessively in facial movement. The over-all effect of facial expression is improved by diminishing

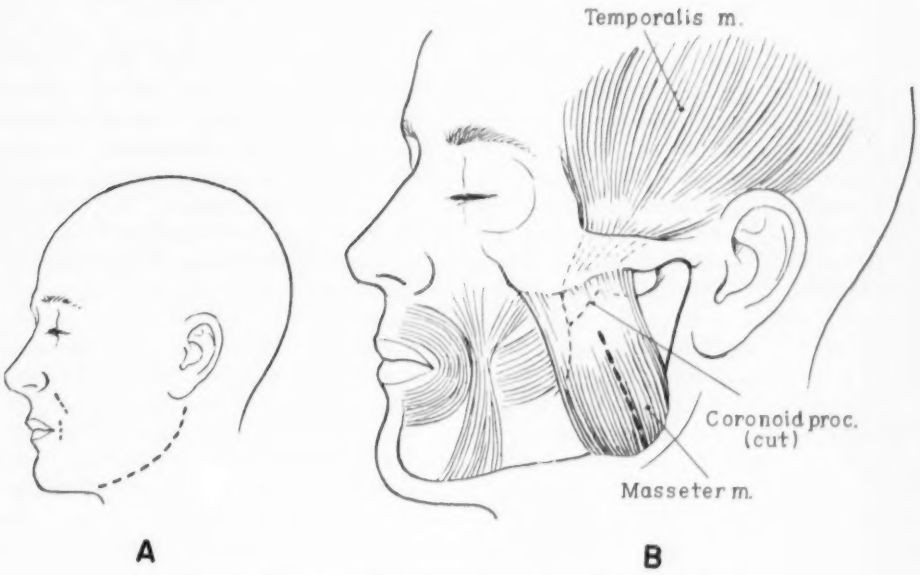
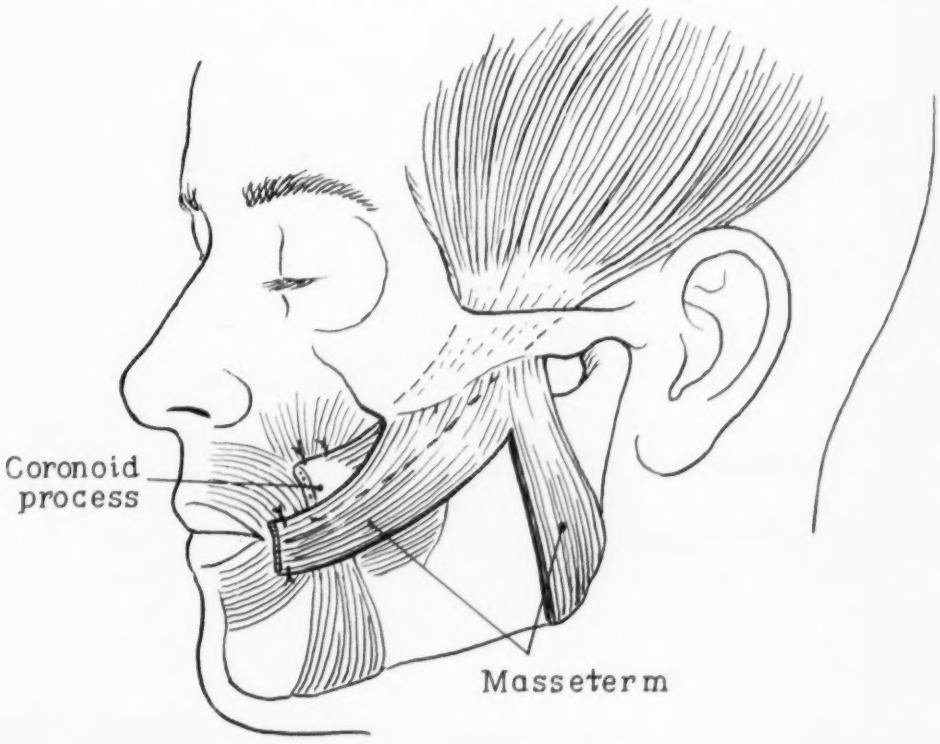


Fig. 2.—Preparing masseter and temporal muscles for rotation.

Fig. 3.—Masseter and temporal muscles repositioned about oral commissure.

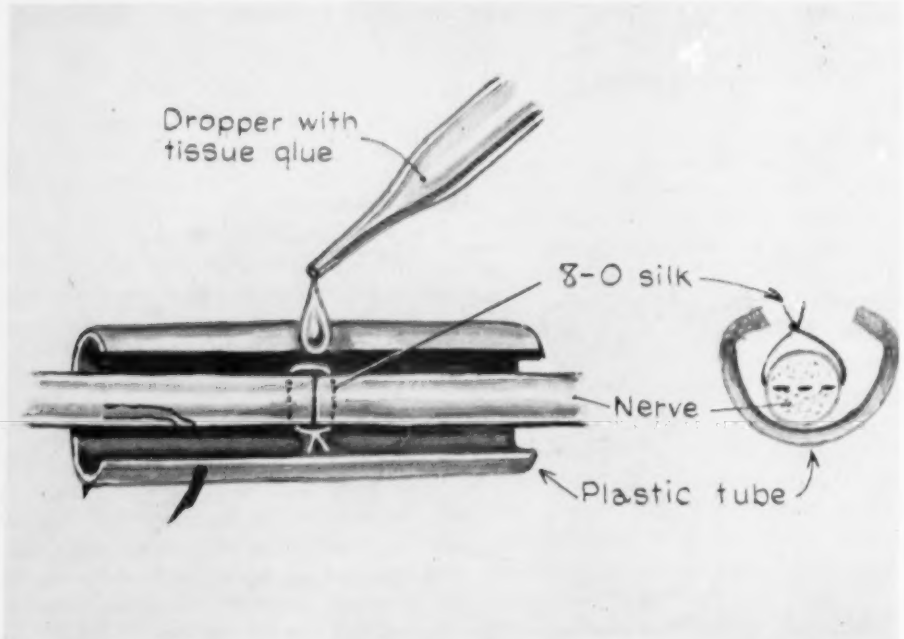


the exaggerated and unbalanced movements of the normal side of the face. This is accomplished by resecting 1 cm. of each of the muscles in the responsible group. A melolabial incision gives adequate exposure to the zygomaticus, levator quadratus, and orbicularis muscles. A segmental resection on the active side and appropriate shortening on the paralyzed side tranquilize the face and establish a quiet and acceptable balance in expression.[3] If only the mandibular branch of the facial nerve is paralyzed on one side, the resultant imbalance is best handled by resecting the mandibular branch on the opposite side, thus restoring symmetry and not interfering with function.

4. Nerve repair. If the facial nerve has been sectioned or a small portion of it resected, the best method of handling the problem is by direct approximation of the cut filaments with 000000 Atraumatic silk. This simple and direct method of repair affords the patient the maximum amount of facial rehabilitation in the shortest possible time. The quality of the return of movement is usually excellent. If direct approximation of all fibers is not possible because of too much loss of certain nerve elements, then indirect approximation, which is the anastomosis of one terminal filament with another terminal filament or subordinate branch, should be carried out. Movement of the face following this type of nerve repair has been seen as early as four weeks postoperatively in a 15-year-old girl, but is usually delayed for a period of five to six months.

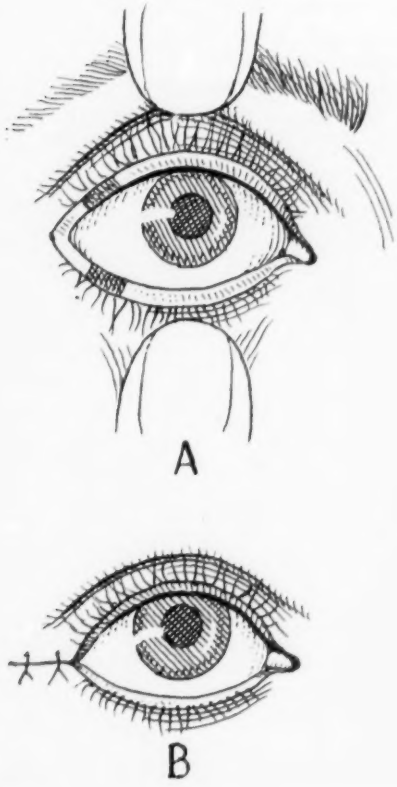
5. Nerve grafting. The substitution of the hypoglossal or spinal accessory nerves for the facial nerve has been used with some success in creating facial movement. It, unfortunately, has no advantage over free nerve grafting and has the disadvantage of either impairing the function of the tongue or the shoulder, and producing an aberrant type of muscle coordination. Free nerve grafting in this instance may consist of the substitution for one or more filaments of the nerve, or the entire nerve itself, depending upon the extensiveness of the

Fig. 4.—Use of plastic-tube trough to stabilize the sutured small nerve filaments in the face.



excisional operation. The great auricular and colli nerves on the contralateral side of the neck are an ideal source of nerve-grafting material. From these branches may be procured partial segments for anastomosis or a complete nerve trunk with four appropriate ramifications. If the facial nerve and its branches have been resected during the excisional stage of the operation, one should routinely and immediately anastomose a free nerve graft. The technique consists of four 000000 Atraumatic silk sutures for the main trunk of the facial nerve and its graft, and one through-and-through 000000 Atraumatic silk suture for the terminal divisions after they have been threaded through a fenestrated plastic tube to maintain the accuracy of their approximation. The anastomosis of these fine filaments may be improved by careful manipulation with a fine probe through the fenestrated plastic tube under high-powered magnification. If the masseter muscle has not been removed during the excisional operation, its anterior third is transposed into the commissure of the mouth and secured there with three mattress sutures. If the masseter muscle is not available, then the temporal muscle and a portion of the coronoid process of the mandible is transposed into the region of the commissure of the mouth.

Fig. 5.—Tarsorrhaphy.



The nerve grafts vary in length and size, depending upon the requirements of the wound. One or several single filaments may be 30 mm. in length, whereas a total facial nerve graft may be 90 mm. in length. Regeneration takes place over a period of 6 to 18 months, depending upon the length of the graft.

The results in these nerve-graft cases are interesting and gratifying. As technical difficulties are overcome and neural chemistry and growth better understood, the outlook will continue to improve, so that one can expect a good return in the majority of instances. The accuracy in the anastomosis in the small filaments in the peripheral part of the nerve bed is always under question. The plastic-tube technique enhances the possibilities of success at this site. One must, however, always be prepared for the invasion of the anastomosis by the ingrowth of scar tissue and also the effect of late scar-tissue contraction about the nerve. The plastic tube should be three times the diameter of the nerve graft so that it cannot cause ischemia of the nerve.

Many interesting experiences developed from this work and are worthy of being related. There is little doubt of the value of an active physiotherapeutic program during the interval of nerve regeneration, which may take as long as 12 months. Galvanic stimulation, active bimanual massage of the cheek, heat, and active movement of the rotated masticatory muscle assist in keeping a high degree of tone in the face and the tissues in a receptive con-

dition for the regeneration of the nerve fibers. Obviously, if the nerve regenerates after a period of 12 months into muscles that have atrophied and fibrosed, one cannot expect good physiological function of that side of the face, even though the regeneration of the nerve graft may be perfect. The regenerated nerve must have the privilege of growing into muscles which have been maintained in good tone and quality.

It was invariable that when function did return to the face the potential for rehabilitation was much greater than the patient recognized. Indeed, some patients had a return of potential function to the face and were not aware of it. A specific program of active exercise of the side of the face that was operated on for a period of two to four weeks is carried out to help the patient reassociate the coordinated movements of the muscles of the face with the higher neural centers. This ordinarily requires considerable concentration and practice. The reassociation of these muscles through their neural pathways with the higher centers is awkward, confusing, and sluggish in the beginning, and indeed never attains the original and natural harmonious activity. Gradually movement is oriented with the normal side, and what was originally a slight twitch progresses to a clumsy type of mass movement and, finally, some refinement of movement appears. This usually requires tens of thousands of practice movements. It is reasonable to assume that when the success is less than 50% the lack of coordination of the neural impulses is probably more significant than the technical failure of the nerve anastomosis.

Hypertonia appeared on the side of the face operated on and was associated with a sensation of tightness and involuntary twitches. This ordinarily subsided as the rehabilitative program progressed. Mass movements were a constant feature but were minimized by training.

A considerable amount of work remains to be done on regeneration of the facial nerve. It has a great capacity for regeneration, perhaps more than the other peripheral motor nerves of the body. When the opportunities for success appear meager, one is often delightfully surprised to find a considerable amount of return of muscular movement. This further emphasizes the regenerative potentialities of this nerve and brings up the question of miotaxis. Spontaneous regeneration of motor nerves in children under the age of 7, where no attempt was made at grafting, has been reported.[2] Its occurrence in adults is questionable, yet one cannot help but feel that it plays a role in some instances. It should not be used as a substitute for reconstructive and rehabilitative nerve grafting.

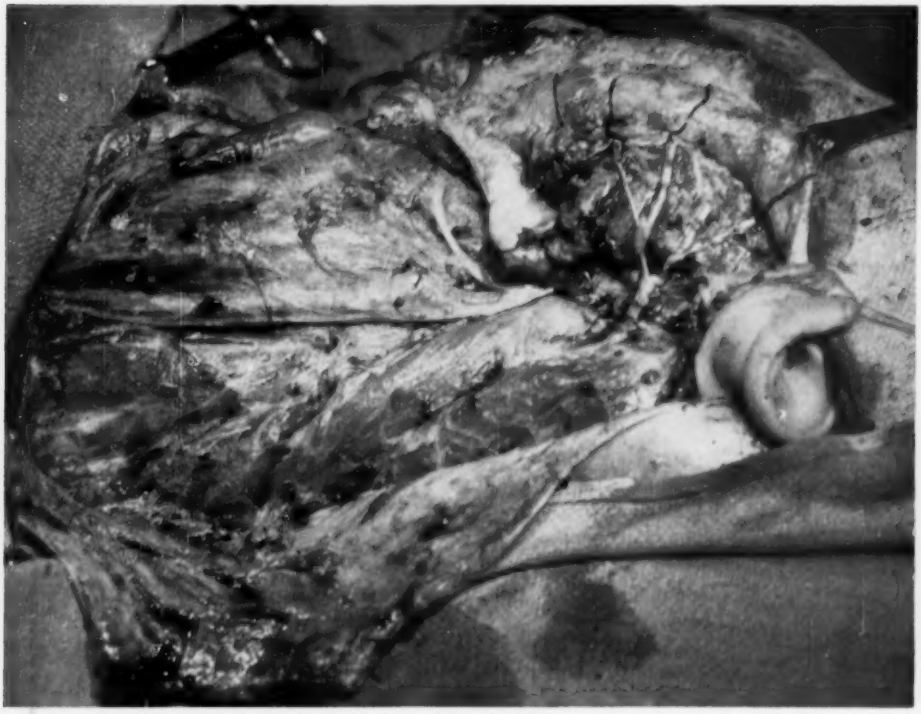
The following case is worthy of brief notation: A 15-year-old girl presented a recurrent high-grade mucoepidermoid cancer that had been previously irradiated and operated on. In resecting the tumor, the main trunk of the facial nerve was transected along with the four principal peripheral branches. A direct reapproximation of the residual nerve fibers was carried out without a free graft. Within a period of five weeks the patient had active and well-

Fig. 6.—Normal facial tone in 15-year-old girl, two months after suturing of facial nerve in five divisions, including the main trunk.

Fig. 7.—Facial-nerve function after resection of the parotid gland, lateral neck, and nerve graft.

Fig. 8.—Autogenous facial supports in right side of face following extensive excision for recurrent adenocarcinoma of parotid gland and neck.

Fig. 9.—Reconstruction of facial nerve by great auricular nerve graft following radical resection of parotid gland and lateral neck for cancer.



coordinated movement of the middle and lower third of the face. This unusual experience would indicate that the brief time interval would have been inadequate for a complete reaction of degeneration and regeneration, and that the interference of the neural pathway was minimal and transitory in character.

Occasionally, upon reexploration of a facial-nerve graft, apparent separation of the nerve filaments at the line of anastomosis seemed to prevail. Upon closer inspection, however, it appeared as if a minimal portion of the anastomosis was intact and the remainder either separated by scar tissue or distorted. Yet, these persons will often have good movement of the face.

In partial resection of the facial nerve (either the temporal or cervical division) residual movement of the face was excellent, emphasizing the multiple network of anastomoses of the different peripheral branches of the facial nerve in the cheek, with duplication of control and innervation. These facts emphasized the situation that it was not necessary for total regeneration of all the nerve fibers to produce good function. If a small percentage of the fibers in the main trunk made key connections in the peripheral apparatus, one could expect good function.

Conclusions

1. The correlation between an accurate microscopic classification and the biology of the tumor is essential for the proper management of tumors in the parotid gland.

2. Rarely is the facial nerve permanently injured in the management of benign tumors of the parotid gland. Many highly malignant tumors, particularly small ones localized in the lateral lobe, may be resected without permanent injury to the facial nerve.

3. In highly malignant tumors involving the major portion of the lateral lobe, or the isthmus or the medial lobe, or those that have already invaded a portion of the facial nerve, radical resection of the entire gland and facial nerve should be carried out. These persons require facial rehabilitation.

4. This rehabilitation consists of immediate nerve repair by direct or indirect approximation, when this is possible.

5. Facial-suspension techniques by employing fascia lata and inert metallic compounds looped through the zygomatic bone or temporal muscle are considered least desirable and are advised only as auxiliary procedures when a more dynamic method is unwarranted or impossible.

6. Restoring facial balance by tranquilizing the muscles of the unoperated side and shortening the muscles of the side operated on effects a marked improvement in facial expression.

7. Muscle-rotation techniques, utilizing the temporal or masseter muscles, offer an excellent type of live physiological suspension and movement associated with masticatory activity.

8. Nerve-grafting techniques, either partial or complete replacement of the facial nerve, are the treatment of choice. In the majority of instances, one can expect a good return of mimetic activity of the face operated on, thus restoring the natural physiological activity of the area operated on.

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Presbycusis

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Presbycusis has been likened to presbyopia, the two conditions having the aging factor in common. In the older population, the age of the person may be ascertained precisely from the amplitude of his accommodation. The aging process of the lens—increase in lipoid content and hardening of nucleus—is physiologic, with an incidence of 100% in humans. In the ear, the hypothesis that there is a decrease with age in the perception of the upper tonal range has been formulated from studies with the Galton whistle, by Zwaardemaker [1]; with the monochord, by Struycken [2]; with the audiometer, by Bunch and Raiford [3] and Montgomery.[4] Other etiologic factors of high-tone deafness include acoustic trauma, infections, and ototoxic drugs. In the differential diagnosis, the universality of the deficiency in perception of the high frequencies in the older age groups is stressed. None of the 60-year old persons in the Bunch and Raiford series heard the extremely high frequencies. Montgomery studied 370 laboratory employees who were healthy people with no history of aural disease. The difference between the composite audiograms of the 20-year old group and those of the 59-year old group was 5 db. for 64 d. v.—2048 d. v. and 25 db. for the 4096 cycle.

A controversy regarding the nature of presbycusis arose at the turn of the century. Zwaardemaker [1] was of the opinion that the lowering of the upper tone limit is a physiologic phenomenon. Sporleder [5] thought that even middle-grade deafness must be produced by factors other than age. Von Stein [6] believed that arteriosclerosis is the cause of presbycusis.

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In Alexander's [7] famous case, histologic changes were found in the internal auditory artery of a case of arteriosclerosis with inner-ear deafness. However, two statistical studies at the Johns Hopkins Hospital indicate that there is no greater incidence of perceptive deafness in persons with arteriosclerosis than in those of comparable age groups without this disease. Bunch [8] reported that 58 patients aged 50-60 years and 55 patients over 60 years, in all of whom the diagnosis of arteriosclerosis was established, showed no significant increase in inner-ear deafness. In Ciocco's [9] series of 1980 cases, the 298 patients with cardiovascular disease showed no greater deterioration of auditory acuity than the others of comparable age groups. In a histologic study by Crowe, Guild, and Polvogt,[10] no parallelism was found between degenerative changes in the neuroepithelium and nerve fibers and sclerotic lesions in the blood vessels of the inner ear.

The recruitment phenomenon distinguishes between lesions of the end-organ and those of the eighth nerve. It is present in Ménière's disease and in acoustic trauma, in which conditions the ganglion cells and afferent nerve fibers are relatively unaffected, the pathology being limited to the end-organs. On the other hand, the nerve-fiber diseases, such as acoustic neurinoma, are devoid of recruitment. In presbycusis, the predominant histologic finding is atrophy of the spiral ganglion cells, according to Saxen.[11] He described, also, another type of presbycusis, which is characterized by degeneration of the epithelial tissues of the cochlea. Schuknecht [12] stresses the fact that in the epithelial atrophy type of presbycusis the loss of hair cells and the loss of spiral ganglion cells are closely parallel. Accord-

ingly, neither the neural atrophy type nor the epithelial atrophy type of presbycusis can show recruitment, as they lack the prerequisite for the production of this phenomenon, which is a near-normal number of ganglion cells. If, however, an old person with high-tone deafness does show recruitment, it may be assumed that his aging has not affected his ganglion cells and that his hearing loss is due to hair-cell damage caused—in the course of his long life—by noise, infection, or drugs.

Frequently, we encounter deafness problems in young and middle-aged persons in whom the audiologic findings suggest a presbycusis pattern. There is also a familial tendency to this type of deafness in some cases. The occurrence in two brothers, two sisters, and in a brother and sister has been witnessed.

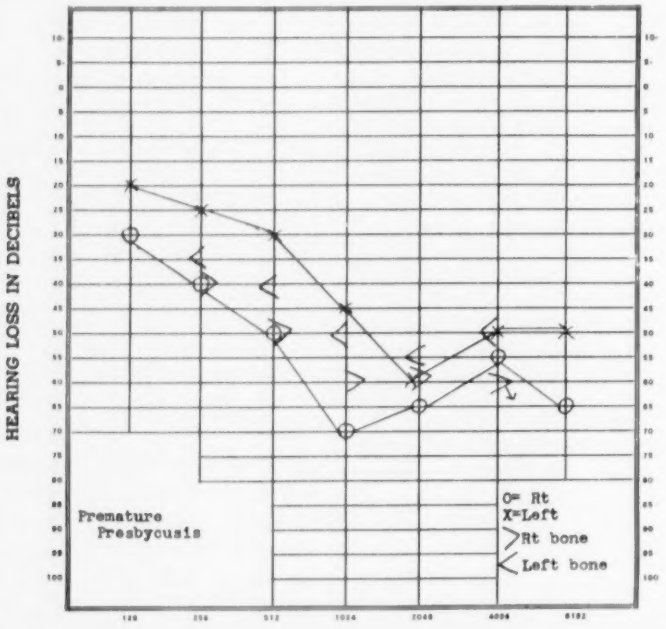
Representative Case

A white man, aged 59, was examined on March 23, 1956. His complaint was the difficulty in understanding conversation at a distance of 5 ft. or more. The several hearing aids he owns are of help only in making sounds at close range louder and "plainer." The onset of the hearing impairment occurred at age 45, and the deterioration has been progressive. The patient is free of tinnitus and never experienced vertigo. He enjoys good health and is a successful professional man. No abnormality was found by otoscopy and nasopharyngoscopy. The Eustachian tubes were patent. There were no abnormal spontaneous neuro-otologic findings, and the caloric test performed on both ears elicited normal responses. The patient's audiogram is shown in the Figure. No recruitment is present. S. R. T. was at the 55- and 35-db. levels in the right ear and the left ear, respectively. The discrimination score was 40% in the right ear and 62% in the left ear. His sister has similar hearing difficulties, and the onset of her progressive deafness also occurred during the middle-age period.

Comment

In this case, premature presbycusis is suggested by the sloping audiogram, progressive perceptive deafness, absence of recruitment, absence of vertigo and tinnitus, poor discrimination score, nonrehabilitability through a hearing aid, and the family history.

The findings in this case conform with the audiologic manifestations of presbycusis



of advanced age, except in regard to slow cerebration, which prevails in the older group. Poor speech intelligibility at a distance of 5-6 ft., in spite of a suitable hearing aid, is a common complaint in presbycusis. This observation was reported by Ersner and me[13] in 1951. A plausible explanation is that the presbycusis ear loses most in the discriminatory advantages of the high consonants. Kerrison[14] states that s and sh sounds carry farthest and can be heard three times as far as f, v, t, th, d, and k. Again, f, v, d, and k are heard farther than b and p. As the high consonants are weakest in sound power, their audibility at a distance is nature's special gift to the normal ear. This special sensitivity is lost in presbycusis. It cannot be remedied by the amplification of a hearing aid, as it is not sound power that counts most. It is the receptive organ that lost its special sensitivity.

As handicapping deafness is present in less than 10% of people aged 60-80 years, it appears that the aging factor alone does not produce the atrophic changes in the inner ear described by Saxen and Schuknecht. Moreover, the occurrence of the presbycusis pattern of deafness in middle-aged people who have no degenerative changes in any other part of the body suggests a specific dystrophy on a nutritional or hormonal basis. Advancing years and heredity constitute predisposing factors.

Summary

A lowering of the upper tone limit is a universal finding in old people.

An otologic entity with a distinctive histopathologic pattern has been described by Saxen and Schuknecht under the term "presbycusis." It is encountered more frequently in advanced years, but it occurs also in the younger age groups. Heredity is a predisposing factor. It presents a characteristic audiologic pattern. It is hypothesized that presbycusis constitutes a specific dystrophy of the inner ear. To deter the progress of the deterioration, the help of the sciences of geriatrics, nutrition, and endocrinology must be sought.

1923 Spruce St. (3).

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

A Delayed Sidetone Test for Detecting Uniaural Functional Deafness

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Functional elements in medicolegal evaluations of hearing disabilities generally exceed those observed in diagnostic or therapeutic examinations. The forensic audiologist charged with the responsibility of measuring the organic deficit in these cases must take particular care to isolate nonorganic components and seek to quantify them.

Clinical experience has shown that functional hypacusia often takes the form of a uniaural loss. There are three major reasons for this fact: The unsophisticated person tends to overevaluate the gains resulting from the demonstration of uniaural deafness, it is often assumed that it is easier to feign accurately from test to retest a total hearing loss in one ear than it is to repeat the thresholds of a binaurally symmetrical loss, and with this kind of hearing loss there need be few gross changes in communication behavior and daily living habits.

The detection of the uniaural type of functional deafness can be important even though disability evaluations for such losses tend to be small. If these functional uniaural losses are accepted as organic, small decreases in the acuity of the allegedly better ear can lead to substantial increases in disability evaluation. Therefore, an objective audiometric test which permits accurate detection of functional deafness of the uniaural type is needed, and must certainly gain in usefulness.

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Present audiometric tests employed to demonstrate organic, one-sided deafness have characteristics which can lead to serious test error or to test failure. A brief discussion of these techniques and their shortcomings as seen by the forensic audiologist is important.

It is most likely that the Stenger tests represent the earliest attempts to develop an audiometric procedure designed solely to detect the uniaural functional hearing loss.[1] In addition, the Lombard,[2] the Weber,[3] spoken and whispered voice,[4] repeated threshold tests,[5] and others have been used to detect one-sided functional deafness. Experience has shown, however, that the latter procedures are not especially well suited to the task set before them because detection of functional elements depends upon the interpretation of subjective responses. These techniques do not permit the examiner to isolate acoustically the ear under test or to minimize errors introduced by the cross-over of sound to the opposite ear. What is needed is a procedure which tests one ear at a time by nullifying the functions of the ear not under test.

Until recently, the Stenger tests have been the most useful means of demonstrating organic uniaural deafness. However, these tests, whether they be given with matched tuning forks or with the elaborate, modern two-channel audiometer, are subject to variation and error because they involve the stimulation of both ears.[6] It is not possible

to isolate the ear under scrutiny so that it can be tested separately without consideration of the threshold of the other ear in these tests.

Although the Stenger tests are widely used, they have characteristics which can lead to spurious test results or even test failure. Primary among these is the degree of subject sophistication, for success depends upon the fact that the subject is uncertain of the principle or administration of the test. If the subject understands the operation of the test, he can respond to the tone presentation so as to give the appearance of a severe loss in the ear under test when in fact the ear is normal.

In addition, there are certain technical problems related to the administration of the test which can provide further sources of test error or failure. Electronic cross-over, or induction, from one stimulus channel to the other can result in test failure by altering the intensity of the stimulus presented to the allegedly better ear. Tone-interrupter switches having slow tonal rise times can produce test failure, for successful administration of the Stenger test depends upon the ability of the audiologist to present tones suddenly at precisely controlled intensities. Noisy or dirty switches which during their operation develop extraneous sounds can lead to spurious test results by indicating to the subject that the tones are being altered, while tone-interrupter switches which have too rapid tonal rise times can lead to test failure by indicating to the subject changes in the tone presentation through acoustic clicks.

Under the most ideal conditions the Stenger test must be done quickly with the most skilled and subtle techniques, for the value of the test tends to deteriorate rapidly after the first series of tone presentations. There appears to be a tendency for even the inexperienced subject to become aware that both ears are being stimulated when the test is prolonged, and thus continue to respond to both stimuli. When this occurs, the procedure is no longer effective.

Interpretation of the Stenger tests is difficult because they are most accurately interpreted during the actual test-administration period. The audiologist often must depend upon minimal responses for the successful interpretation of the subject's reactions to the tone presentations, reactions difficult to describe or record.

The Stenger tests can often fail in their clinical application because they are dependent upon subjective reactions. If the subject cannot or will not cooperate fully with the audiologist, the test results in failure. Consequently, a negative Stenger test should never be looked upon as conclusive proof of organicity. Such test results must always be corroborated through the use of other clinical procedures.

Although demonstration of a shadow curve can corroborate a negative Stenger test result,[7] this technique has value only when the threshold of the good ear is better than 30 db., or when the organic threshold differential between ears is 60-70 db. Whenever these conditions are not met, inconclusive test results can obtain. This limiting characteristic makes it necessary to rely upon other tests in order to substantiate smaller organic threshold differentials between ears.

Tests for the detection of uniaural functional deafness depending for their operation upon the sound-localizing properties of binaural hearing[8] can often be used to support a negative Stenger test, particularly in the case of the unsophisticated subject. However, certain problems related to the administration of such tests or the psychoacoustical properties of one-sided organic deafness can lead to test failure. It has been noted in certain cases of long-standing uniaural organic deafness that the ability to localize sound laterally can be seriously impaired. No matter which ear receives the higher relative sound intensity, the sound is perceived in the better ear or at midline. The audiologist must recognize the possibility of such responses and under no circumstances interpret them as being indic-

ative of complete deafness in the allegedly poorer ear without supportive evidence, especially in cases of suspected congenital or long-standing loss.

More recently, because of the increasing need for additional procedures capable of detecting the uniaural functional hearing loss, other tests which might be termed "tests of confusion" [9] have been developed. The success of these procedures depends upon the ability of the audiologist to manipulate the piecemeal presentation of the segments of a short story or anecdote in such a manner as to make it difficult for the subject to remember exactly which portion of the story was presented to each ear. Confusion can be revealed by asking the subject a series of questions whose answers must be based upon what was heard. If there is a functional deficit in one ear, this fact may be indicated by the answers to the questions. The limitations of such a technique are obvious. The sophisticated subject may be able to avoid detection of a uniaural functional loss by giving evasive or misdirecting answers, while the less sophisticated subject may state that he can remember nothing of what was told to him. In either case, the result is test failure.

The preceding discussion, although not definitive, makes it clear that there is further need for additional tests of uniaural functional deafness, tests which are dependent upon the application of sound psychoacoustical principles rather than psychological tricks or subject confusion. Although modification of the Doerfler-Stewart test,[10] and the PGSR procedure [11] has been of value in the detection of uniaural functional deafness, certain technical problems relating to the matter of nullifying the functions of the ear not under test still have not been solved.

Earlier study has indicated that delayed sidetone testing of separate ears is feasible and can be accomplished through auditory masking of the ear not under test.[12] This procedure makes it possible to isolate acoustically the ear under test by nullifying the functions of the opposite ear. Further, extensive use of delayed sidetone audiometric techniques has emphasized the highly desirable objective nature of this procedure. This objectivity has been clearly demonstrated during the exploratory application of delayed sidetone techniques in a wide variety of clinical cases wherein only a very small percentage failed to exhibit easily discernible manifestations of interference with the subject's normal speech processes.

The present investigation involves a modification of earlier testing techniques which were applied to normally hearing subjects, and is concerned with the clinical validation of an audiometric procedure designed to detect the presence of uniaural functional deafness.

Subjects

Seventy veterans with medically diagnosed organic uniaural hearing losses were subjected to exhaustive audiometric study in order to substantiate and quantify their aural deficits. These veterans were seen for adjudicative purposes or for medical treatment processing in the Audiology and Speech Correction Clinic, Veterans Administration Regional Office, Los Angeles. No subject showing any degree of neuropsychiatric disorder or any evidence of a speech or language disturbance, in addition to the uniaural hearing loss, was included in the study. The following audiometric criteria for subject selection were observed:

1. No subject having less than a 40 db. threshold differential between ears or a speech reception threshold worse than 15 db. for the better ear was studied.

2. No subject with a speech discrimination score of less than 90% for the better ear was selected for study.

3. No subject with a pure-tone loss exceeding 20 db. for any single frequency in the speech range of the better ear was selected.

Thirty-seven subjects showed the greater loss in the right ear, and thirty-three showed the greater loss in the left ear. The losses in the poorer ear ranged from 40 db. to no response on standard audiometric equip-

ment, with a mean differential between ears of 69.7 db. These subjects were selected from the routine clinical caseload of more than 1000 veterans a year undergoing medicolegal evaluation.

Equipment

A modified Magnacorder Tape Recorder, Model PT-63AH with PT-63J amplifier, provided the delayed sidetone. Generation of the saw-tooth masking noise and control of the intensity levels for delayed sidetone and masking noise were done through the use of a U. S. Recording Hearing Evaluator, Model 200. This dual channel equipment made it possible to present at designated sensation levels the masking noise and the delayed sidetone to the subjects through Permaflux receivers, Model PDR-8 with MX-41-AR cushions. The auditory testing equipment was located in a standard two-room, acoustically isolated test suite, and was aligned and calibrated at regular intervals.

The standard prose reading passage used in all tests was taken from a fifth-grade reader currently employed in the Los Angeles public schools. Reading materials at this level were selected to ensure that few subjects would be rejected for defective reading. This reading selection, subsequently titled the "Kitty Hawk" passage, contained 500 syllables of expository prose which had been demonstrated to be uniform throughout with respect to reading difficulty.

Research Procedures

The total population of 70 uniaurally deafened veterans was tested under two different delayed sidetone conditions. As they appeared for routine audiometric testing, the veterans were assigned alternately to one of two test sequences, each of which involved the two different test conditions shown in Table 1.

Thus, for test sequence MP_1, the subjects first received a 60 db. sensation level of delayed sidetone directed to the better ear, while the poorer ear was masked by a saw-tooth wave-form masking noise presented at 80 db. re normal threshold for speech. The standard prose passage was read aloud by the subject and the elapsed reading time recorded. After a suitable rest period, the test conditions were reversed and the subject was instructed to reread the same passage. The two reading sessions constituted all the testing done with each subject in test sequence MP_1.

For test sequence MB_1, the test presentation order was changed so that each subject in this group first read the common test passage with the masking noise to the better ear while the poorer ear received the delayed sidetone. Again, after the passage had been read and the elapsed time recorded, the test conditions were reversed and the elapsed reading time for this reading session recorded.

Results

The testing procedures described above resulted in two elapsed oral reading times for each subject. These reading times were designated "MP" (masking noise to the poorer ear) and "MB" (masking noise to the better ear). Table 2 summarizes data obtained from the two distributions of oral reading times secured from the present investigation and one distribution of oral reading times resulting from a previous delayed sidetone study involving binaural presentation to normally hearing subjects, which was designated condition A.

Reference to Table 2 reveals that there was a 36.4-second mean difference between

TABLE 1.—*Test Conditions*

Test Conditions	Code	Sensation Levels		Sidetone, Delay, Sec.
		Better Ear	Poorer Ear	
Delayed sidetone to better ear, masking to poorer	MP	60 db. sidetone	80 db. masking	0.16
Masking to better ear, delayed sidetone to poorer	MB	80 db. masking	60 db. sidetone	0.16

TABLE 2.—*Grouped Reading Times in Seconds*

Test Condition	N	Mean Age, Yr.	Males, %	Mean Times	Standard Deviations	Ranges
MP-masking to poorer ear	70	36.5	95.7	178.3±5.2	42.9±3.7	117.8-309.2 (191.4)
MB-masking to better ear	70	36.5	95.7	141.9±3.1	25.6±2.2	92.0-238.6 (146.6)
Binaural presentation	40	37.6	75.0	179.8±3.8	24.4±2.8	130.6-238.2 (107.6)

TABLE 3.—*Analysis of Variance Between Test Conditions MP and MB*

Conditions Compared	Sums of Squares	Degrees of Freedom	Mean Squares	F-Ratio	P
MP-MB	464.5 1770.5	1 138	464.5 12.8	36.3	>0.01

test condition MP and test condition MB. In order to test the significance of this difference an analysis of variance was computed (see Table 3), and the resulting F-score indicated that the variance between test conditions MP and MB was very significant.

Next, analyses of variance were obtained by comparing the two test conditions of the present investigation with the test condition previously used in a delayed sidetone study of normally hearing subjects. This test condition involved reading the same 500-syllable passage used in the present study under a 60-db. sensation level of delayed sidetone binaurally presented. Table 2 summarizes data resulting from the analysis of test times obtained from the previous test condition. Analyses of variance scores were computed by comparing condition A with conditions MP and MB. Table 4 summarizes the details of these computations.

Reference to Table 4 makes it clear that the variance between test conditions MP (masking to the poorer ear) and A (binaurally presented delayed sidetone) is not significant. On the other hand, the variance between condition MB (masking noise to the better ear) and the same condition A was very significant.

During the testing it was seen that one test sequence might be more effective than the other. In order to determine which test sequence was the more effective, the total subject population of 70 veterans was divided into two equal groups. This division was made on the basis of the ear receiving the delayed sidetone stimulation first. Table 5 illustrates the division of the population and the details of the test presentation sequences. This division of the subjects resulted in four elapsed reading times, which were analyzed in terms of the intergroup differences. Table 6 summarizes data derived from the four distributions of reading times.

Reference to Table 6 reveals that different sequences of presentation resulted in different mean reading times. It was necessary, therefore, to determine whether or not these differences were statistically significant. Analyses of variance for the four reading time distributions were computed. Table 7 contains details of the computations of F-scores which quantified the degree of variance between the distributions of reading scores as they were paired in various combinations.

The F-scores resulting from the above computations revealed that all the paired combinations of group reading times were

TABLE 4.—*Analyses of Variance Between Clinical and Normal Groups*

Conditions Compared	Sums of Squares	Degrees of Freedom	Mean Squares	F-Ratios	P
MP-A	16.2 1553.2	1 108	16.2 14.4	1.125	Nonsig.
MB-A	2234.1 696.9	1 108	2234.1 6.453	346.21	>0.01

TABLE 5.—*Test Presentation Sequences*

Order	Conditions		
	1	MP	MB
	2	MP_2	MB_2

TABLE 6.—*Group Reading Times in Seconds*

Test Orders	N	Mean Times	SD	Range
MP_1	35	171.5±6.0	35.2±4.3	117.8-298.3 (180.5)
MP_2	35	134.1±6.9	40.1±4.9	92.0-238.6 (146.6)
MB_1	35	151.4±3.4	19.8±2.4	106.4-236.7 (130.3)
MB_2	35	182.7±5.5	31.8±3.9	127.6-309.2 (181.6)

significantly different. This result was taken to mean that presentation order can have a marked influence upon the difference between contrasted test conditions. Because of this fact it became necessary to establish which test sequence produced the larger temporal difference between the two test conditions. In order to do this, mean differences between the reading times resulting from the two test sequences were obtained. Table 8 presents these mean differences. The table suggests that test sequence MP_1-MP_2 is the more effective test presentation order because it yields the greater mean time difference. Further evidence of the greater effectiveness of MP_1-MP_2 is contained in Table 9, which presents the scatter and range of test time differences and the number of test time differences falling below 10 seconds. This table makes it clear that test presentation sequence MP_1-MP_2 is more effective because the range of test time differences was smaller, the distribution was more closely grouped, and only one test time difference was below 10 seconds.

Next, attempts were made to establish whether or not a pattern existed between the extent of the difference in acuity between ears and the mean difference in the two over-all test conditions MP and MB in seconds.

Table 10 indicates that no discernible relationship exists between the extent of the difference in acuity between ears and the mean time differences between the two test conditions.

Finally, the time differences between the two test conditions (see Table 9) were examined in order to establish a rationale for setting up a minimal standard below which a functional involvement in the poorer ear must be suspected. Ten seconds was chosen as the screening level for possible functional elements.

TABLE 7.—*Analyses of Variance for Testing Differences Between Test Presentation Sequences*

Conditions Compared	Sums of Squares	d. f.	Mean Squares	F-Ratios	P
MP_1-MP_2	1844.4	1	1844.4		
	2705.4	68	39.8	46.3	>0.01
MP_1-MB_1	1358.4	1	1358.4		
	2007.9	68	29.5	46.0	>0.01
MP_2-MB_2	471.1	1	471.1		
	1700.9	68	25.0	18.8	>0.01
MB_1-MB_2	848.8	1	848.8		
	1981.6	68	29.1	29.1	>0.01
MB_1-MP_2	2527.6	1	2527.6		
	2986.1	68	43.9	57.6	>0.01
MP_2-MB_2	1241.4	1	1241.4		
	2679.1	68	39.4	31.5	>0.01

TABLE 8.—*Mean Time Differences Between Test Sequences*

Sequences Compared	Mean Differences, Sec.
$MP_1 - MP_2$	37.4
$MB_1 - MB_2$	31.3
Difference	6.1

Comment and Conclusions

This investigation involved the clinical validation of an objective audiometric pro-

TABLE 9.—*Grouped Time Differences Between Test Conditions in Seconds*

Times	$MB_1 - MB_2$	$MP_1 - MP_2$
≥ 0	1	0
0 — 9	5	1
10 — 19	9	6
20 — 29	8	10
30 — 39	2	5
40 — 49	1	3
50 — 59	0	4
60 — 69	3	3
70 — 79	2	1
80 — 89	0	1
90 — 99	1	1
100 — 109	2	0
110 — 119	1	0

cedure designed to detect uniaural functional deafness. The results of the study demonstrated that this testing procedure can effectively detect the existence of a uniaural functional hearing loss, and that its utilization in a comprehensive audiometric examination is easily accomplished.

Conclusions reached in previous research which asserted that each case must be evaluated in terms of the subject's own reading performances were substantiated. Because of the wide range of normal reading rates encountered among subjects in the usual test situation, it became clear that the critical detection index for this testing procedure must involve the temporal differences in delayed sidetone involvement between the good ear and the impaired ear. Conversely, if there is no difference in delayed sidetone involvement between the two test conditions used, or if the second reading time is shorter than the first, then there is clear indication that a functional loss exists.

One test presentation sequence was superior to the other, for larger time differences were obtained when the delayed sidetone was presented first to the normal ear and then to the impaired ear. This occurred in spite of any perseveration of the delayed sidetone interference with speech during the testing of the impaired ear.[13]

TABLE 10.—*Grouped Differences Between Ears and Mean Time Differences Between Test Conditions MP and MB*

Difference Between Ears, Db.	Mean Time Differences, Sec.	N
40–49	27.6	20
50–59	30.2	11
60–69	19.3	1
70–79	20.2	6
80–89	62.4	11
90–99	51.4	17
100+	14.8	4

Out of the 70 subjects tested 1 case presented atypical results wherein the reading time for the passage with delayed sidetone to the better ear was shorter than the time for delayed sidetone to the poorer ear. This subject was a high-school graduate and did not have any particular difficulty with the fifth-grade reading material. Extensive testing before and after delayed sidestone presentation indicated that the uniaural loss in this case was purely organic in nature. It should be noted that this atypical performance resulted from the test sequence which proved to be the less effective. However, this subject displayed abnormal speech production during the test condition involving presentation of delayed sidetone to the better ear and no speech abnormalities during the test condition wherein delayed sidetone was presented to the poorer ear.

This exception to the usual test results emphasizes the need for comprehensive medicolegal management of this type of case. Each case being presented for medicolegal evaluation of a hearing disability should be considered a priori as possessing a possible functional component. The existence of every organic uniaural hearing loss must be corroborated by assessment of prior vocational and/or military experience, an otolaryngological examination and diagnosis, and a review of social and psychiatric records. In addition, the appraisal of all audiological records, particularly those concerned with hearing tests showing variations, is necessary in order to establish a previous organic threshold base line.

Administration of the Test

All uniaural hearing losses demonstrated by standard audiometry should undergo testing designed to detect any functional component. This is imperative in the following cases: sudden increases in the hearing loss, losses without apparent etiology,

losses showing variations in previous audiograms, and cases not demonstrating auditory behavior characteristic of the uniaural loss. The present study has shown that a clinical test utilizing delayed sidetone testing techniques can very effectively detect functional uniaural deficits, and that the test in question should be administered in the following manner:

Step 1. Obtain organic thresholds, including speech reception threshold for the better ear.

Step 2. Screen the subject to establish his ability to read at or above the level of the material used.

Step 3. Give instructions regarding the conduct of the test. Care should be taken that the subject understands what is expected of him, especially that he is not to stop or hesitate while reading.

Step 4. Present a sensation level of 60 db. of delayed sidetone to the better ear while at the same time presenting a masking noise level of 80 db. re normal threshold to the apparently poorer ear.

Step 5. Signal the subject to begin reading the test material which should consist of at least 500 syllables of easily read expository prose. The elapsed reading time should be measured, preferably through the use of a stop watch.

Step 6. Reverse the above conditions so that the same level of delayed sidetone is presented to the apparently poorer ear, while masking noise at 80 db. re normal threshold is presented to the better ear.

Step 7. Signal the subject to begin reading the same test passage used previously; elapsed reading time should be measured as before.

The objective nature of this test makes its interpretation a simple matter. In the event that the time difference is less than 10 seconds, a functional element in the allegedly poorer ear is indicated. A difference of more than 10 seconds between reading times is strongly indicative of an organic uniaural deficit, although this finding should not be used to quantify the exact threshold differential between ears.

Dramatic differences in speech production between the two test conditions can also suggest a difference in thresholds, even in the small percentage of cases presenting time differences of less than 10 seconds. On the other hand, when the time difference is small and no difference in speech production exists between the two test conditions, a functional component in the allegedly poorer ear must be suspected.

Summary

Seventy cases of uniaural organic deafness were studied in order to validate a new, objective delayed sidetone test designed to detect uniaural functional deafness. The investigation resulted in the following findings:

1. Delayed sidetone testing techniques can detect a uniaural functional deficit.

2. Clinical utilization of these testing techniques is feasible in standard audiology clinics.

3. The results of the testing technique are objective, and their interpretation is easily achieved. Specific instructions for presenting the delayed sidetone test are given.

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The Relation of Occupational Noise Exposure to Loss of Hearing Acuity

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One of the biggest obstacles in the way of an adequate statistical study of the effects of occupational noise on hearing acuity has been the scarcity of reliable long-term audiometric observations on industrial personnel. The effect of very high noise levels can be detected in a short period of time, as illustrated by McCoy's [1] one-year study of chipping workers, but knowledge of the effects of lower noise levels requires proportionately longer periods of observation. Lacking satisfactory follow-up data, many investigators [2-7] have been compelled to draw inferences on the basis of current audiograms taken on noise-exposed workers, using as control data the audiograms of other employees in the plant or the findings of large-scale population surveys.

This method of study has two drawbacks. First, the investigator must take into account the amount of hearing loss that has been incurred as a result of various types of acoustic trauma prior to the occupational noise exposure that is being investigated, e. g., noise exposure in a previous job, gunfire noise in the military service or in recreational activities, and miscellaneous acoustic trauma. These kinds of noise stresses would not introduce bias into the findings of the study if the amount of such exposure was distributed randomly between the exposed and control groups, but such an assumption would be difficult to validate.

The second disadvantage arises from the fact that, ordinarily, employees have been exposed to the noise in their work area for different periods of time, so that the analysis becomes further complicated by the problem of taking into account duration of exposure.

The two variables, preexposure acoustic trauma and duration of current occupational exposure, can be controlled when the following type of longitudinal study design is employed.

At a given point in time, let us say, year x, measure the hearing acuity of a group of workers exposed to various levels of occupational noise stress. After a period of k years has elapsed, measure their hearing acuity again. Then compute the change in hearing acuity that has occurred between year x and the year $x+k$. The effects of the occupational noise to which the workers have been exposed between years x and $x + k$ can then be analyzed by studying the relationship of specifically defined noise levels to changes in hearing acuity, taking into account the age and degree of hearing loss of each subject at year x. Thus, noise stress prior to year x does not affect the analysis of the data, and the duration of exposure is held constant, i. e., at k years.

Audiometric data for a study of this type were made available by a plant employing 1318 men and 131 women. Since 1947, it has been the practice of the medical section to give an audiogram to every employee each time he appears for an annual physical examination, regardless of his occupation or the noise level in his work environment. Thus, the testing of all employees provided control and exposed groups from the same population.

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TABLE 1.—*Distribution of 1049 Male Employees Studied, by Present Age and Occupation*

Occupation	Present Age, Yr.								Total
	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	
Professional and technical	8	9	10	31	15	7	14	10	104
Managers and supervisors	0	3	6	14	13	9	13	13	71
Clerical workers	8	11	13	6	11	9	11	0	69
Craftsmen	20	83	106	106	71	63	70	52	571
Operatives	9	24	36	45	39	27	18	20	218
Service workers	0	0	0	1	2	2	1	1	7
Laborers	0	1	1	0	0	1	5	1	9
Total	45	131	172	203	151	118	132	97	1049

The analysis of the effects of the plant noise is based on the changes in hearing acuity that occurred during a five-year period. For the purposes of this study, the period of observation on each employee is approximately the five years that follow the date on which the first audiogram was taken. For most workers, this period extends from 1947 or 1948 through 1952 or 1953.

Nature of the Data

The Employees.—Table 1 shows the occupation and age distribution of the employees included in the study. The plant contains a large proportion of older workers with long periods of service. This group predominates because of the seniority system in layoffs and low labor turnover brought about, to a large extent, by company policies that reward long-term employment. With respect to occupations, the plant consists primarily of skilled and semiskilled workers, but it also contains a large number of persons who fall into professional, managerial, and clerical categories. Employee stability and diversity of occupations are characteristics of the plant's labor force that render it favorable to the requirements of this study.

The Noise Environment.—In almost all of the areas in the plant, the noise is of the continuous, steady-state type, rather than intermittent or impact, and ranges in intensity from about 60 db. in the clerical and administrative locations to about 90 db. in the noisiest manufacturing areas. For purposes of this study, the employees were placed into one of three noise classifications: (*a*) less than 75 db., (*b*) 75 to 84 db., and (*c*) 85 to 90 db. The over-all noise level in each area was determined by readings taken at the center of the noisiest section in the area and at the operator's position at a machine. Employees were placed into a noise category according to the area in which they worked.

Since the higher frequencies are known to produce greater auditory damage than equivalent intensities in the lower frequencies, descriptions of occupational noise should include noise spectra as well as over-all intensity levels. In this study, however, the use of octave-band analyses was not feasible. A large variety of machines take part in the manufacturing operations, so that there are a great many different noise spectra to which the employees are exposed, even within one work area. Consequently, it is very difficult to describe the daily noise exposure of any given worker in terms of specific intensities in each octave band.

Although practical considerations preclude the use of noise spectra in this study, the three over-all noise categories do represent three distinct levels of noise stress, and, as such, can provide valuable information.

The number of employees that fall into each noise classification is shown in Table 2. Intensities less than 75 db. are not considered noise stress. Persons exposed to noise in this category are used as controls, and their changes in hearing acuity are assumed to be the result of presbycusis.

The Audiograms.—The audiograms were taken with a Western Electric 6 type of audiometer by one technician throughout the study period. The tones included in the audiogram were 128, 256, 512, 1024, 2048,

TABLE 2.—*Noise Exposure of 1049 Male Employees by Age at Onset of Observation and Stage of High-Frequency Hearing Loss at Onset of Observation (Initial Stage)*

Age at Onset of Observation, Yr.	Initial Stage	Noise Exposure			Total
		Less than 75 Db.	75–84 Db.	85–90 Db.	
20–34	0	60	41	53	154
	1	37	45	44	126
	2	33	36	49	118
	3	0	1	4	5
Total		130	123	150	403
35–44	0	42	26	22	90
	1	35	32	41	108
	2	41	38	47	126
	3	5	5	4	14
Total		123	101	114	338
45–59	0	13	10	5	28
	1	39	29	30	98
	2	44	55	43	142
	3	10	18	12	40
Total		106	112	90	308
Grand Total		359	336	354	1049

4096, and 8192 cps. The hearing loss for each was obtained by gradually decreasing the intensity of the tone until it fell below threshold, and then increasing the intensity until it reached threshold again. The tone was sounded intermittently, and the subject, facing away from the technician, responded by raising his hand. The threshold determinations were recorded graphically on a card furnished by the Western Electric Company for this purpose.

The testing room was quiet, except for a low-frequency hum. An octave-band analysis of the noise in the room indicated the possibility that the masking effect of the ambient noise may have introduced slight errors in measuring hearing thresholds of the low frequencies. The present study, however, is primarily concerned with hearing impairment in the high frequencies, so that any audiometric errors resulting from testing-room noise would have little, if any, effect on the findings.

Classification of Audiograms

The term "high-frequency hearing loss" is used here to designate loss of function of certain cochlear nerve receptors, brought about by aging, acoustic trauma, or a combination of both.* This type of hearing impairment begins with a moderate loss of acuity in the high tones, usually in the region of 4000 cps, followed by a more pronounced high-frequency loss, then gradual progression toward the lower frequencies, and, finally, loss of normal hearing thresholds throughout the entire audible range. In measuring hearing acuity for the purposes of this study, the sequence just described has been divided into four stages. Criteria for each stage are described in the following system of classification of audiogram patterns. Illustrative patterns are shown in Figure 1.

These criteria are based on threshold determinations for the frequencies 250, 500, 1000, 2000, 4000, and 8000 cps. The degree of hearing loss for each of these tones is defined as follows:

None	—10 to 20 db., inclusive
Moderate	25 to 40 db., inclusive
Severe	45 db. or more

Stage 0: Normal.—No hearing loss from 250 to 8000 cps, inclusive; that is, no threshold in any of the tones tested exceeds 20 db.

* High-frequency hearing impairment may arise from factors other than aging and acoustic trauma, such as syphilis, arteriosclerosis, measles, head trauma, infections, and the toxic effect of certain drugs. Furthermore, off-the-job noise may have affected the hearing acuity of some of the workers during their period of observation. Practical considerations precluded investigation of these factors, and it is assumed their occurrence, if any, during the five-year study period was randomly distributed among the three noise level groups.

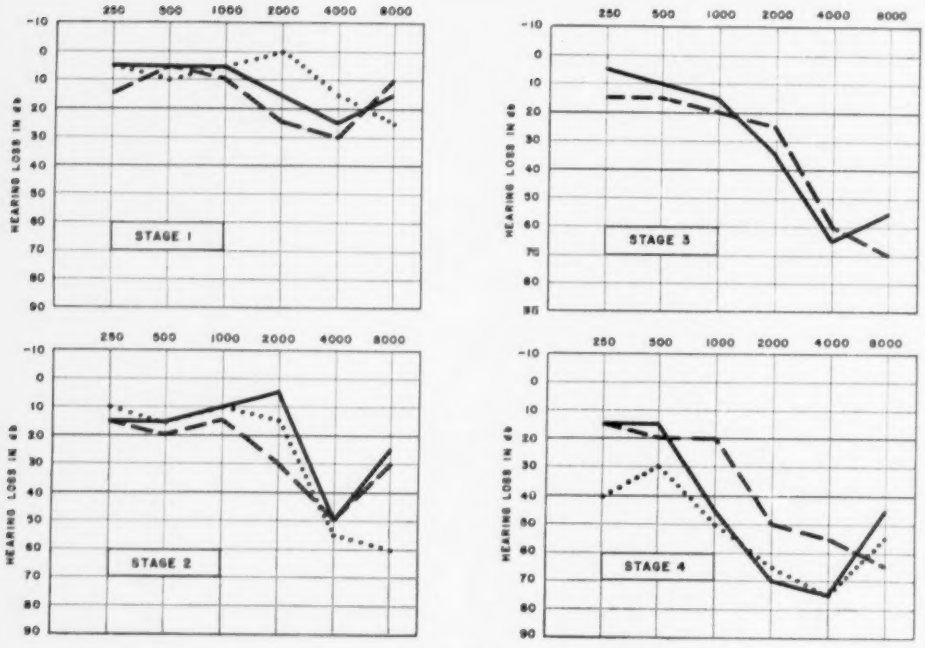

Fig. 1.—Stages of high-frequency hearing loss: illustrative patterns. The various lines shown in each of the four graphs illustrate different audiometric patterns that may be classified according to the indicated stage.

Stage 1: Moderate High-Frequency Loss. No hearing loss from 250 to 2000 cps, inclusive; moderate hearing loss at 4000 cps, 8000 cps, or at both frequencies.

Stage 2: Severe High-Frequency Loss.—No hearing loss from 250 to 2000 cps, inclusive; severe loss at 4000 cps, 8000 cps, or at both frequencies.

Stage 3: Severe High-Frequency Loss with Some Involvement of the Lower Frequencies.—No hearing loss at 250 to 500 cps; no or moderate loss at 1000 cps; moderate loss at 2000 cps; severe loss at 4000 cps, 8000 cps, or at both frequencies.

Stage 4: Severe High-Frequency Loss with Complete Involvement of the Lower Frequencies.—No, moderate, or severe loss from 250 to 1000 cps, inclusive; severe loss from 2000 to 8000 cps, inclusive.

The designation "mixed" is assigned to audiograms which suggest the presence of a conductive type of hearing impairment, with or without perceptive deafness. They show a moderate or severe loss in the lower frequencies (250 to 2000 cps, inclusive), while the degree of hearing loss in the higher frequencies may range from none to severe. Graphically, the patterns are generally flat in appearance, or they may be flat in the lower frequencies only and rise in the higher frequencies.

Criteria for Inclusion in the Study

The 131 female employees were omitted from the present investigation. Of the 1318 males, the audiograms of 1049 were selected for analysis. The 269 male employees who are not in the study were excluded because the noise in their work environment was intermittent or of the impact type, or because their audiograms present one or more of the following conditions.

1. Evidence of middle-ear deafness. The presence of a conductive type of hearing loss was inferred from the audiogram when all thresholds for tones in the lower frequencies (256 to 2048 cps, inclusive) exceeded 20 db. This condition excludes

all persons whose audiogram at the beginning of the study period was classified as "mixed," or Stage 4.

2. Less than five years of observation.

3. Five more years of observation but no audiogram available in the fifth year.

Thus, the study is confined to male employees for whom audiograms are available over a five-year period, who were exposed to a steady, continuous type of noise, whose hearing loss, if any, is assumed to be neural, and whose stage of high-frequency hearing loss at the time of the first audiogram was either 0, 1, 2, or 3.

Measuring Change in Hearing Acuity

The change in hearing acuity that has occurred during the five-year study period is the difference between the stage of high-frequency hearing loss of the worse ear at the onset of the period of observation, as indicated by the first audiogram, and the stage of high-frequency hearing loss of the same ear, as indicated by the audiogram taken at the end of the period.

The audiogram patterns of most employees during the period of observation show a high degree of consistency, but the threshold levels vary in such a way that some employees appear to be at a less advanced stage of high-frequency hearing loss at the end of the period than they were at the beginning. Since nerve-type hearing loss is known to be irreversible, a shift of this kind is not indicative of a real change in hearing acuity.

Some of the reversions may be explained by the presence of auditory fatigue at the time the first audiogram was taken. The extent of temporary threshold shifts produced by the noise in the plant and the rate of recovery time are not known, but investigations into this problem suggest that temporary hearing loss accounts for only a small part of the variation observed in this set of audiograms. Sataloff [8] found no significant auditory fatigue among workers 15 minutes after they were exposed to jet-engine noise whose over-all level was about 106 to 108 db. In his previously cited study,[5] Johnston tested a group of subjects twice. The first audiogram was given 20 to 30 minutes after exposure, and the second, after a 12-day rest. Comparisons between the first and second tests showed no significant differences in hearing loss at 2896, 4098, and 5792 cps. These findings are important in view of the fact that the employees in this study usually took their audiograms 15 to 30 minutes after leaving their work area.

Cox and his co-workers [9] noted almost complete recovery at 4000 cps 43 hours after exposure to noise of about 85 db. per octave band. An average recovery of 8 db. at 4000 cps was found 42 hours after exposure to noise of about 95 db. per octave band. This noise level is greater than the highest noise levels in the plant now being studied. Davis and his co-workers [10] observed that 4000 cps is most susceptible to auditory fatigue and recovers at the slowest rate. They also found that recovery is rapid at first and then slows down, suggesting the possibility that only a small proportion of the temporary hearing loss is present by the time the employee takes his audiogram.

The amount of auditory fatigue is probably related to the noise level that produces it, and can be very great after exposure to 120 db. or more. The studies just cited do suggest, however, that noise conditions as they exist at this plant produce temporary hearing loss that is not large.

The problem of reversions is related to the general problem of the reliability of the audiograms. In a study done by Hughson and Witting,[11] it was found that the error in threshold determinations under ideal conditions is expected to be about ±3 to 5 db. Because of the rigid criteria established for each stage of high-frequency hearing loss, this inherent error alone can account for a reversion to a less advanced stage or a progression to a more advanced stage. A threshold change at 4000 cps, for example, from 25 to 20 db. can mean a change in audiogram classification from Stage 1 to

Stage 0. On the other hand, a threshold change at the same tone from 40 to 45 db. can mean a shift from Stage 1 to Stage 2. Reversions and progressions resulting from audiometric error could be reduced by a coarser classification of stages of high-frequency hearing loss. This could be done, for example, by combining Stages 1 and 2 and Stages 3 and 4, so that there would be three instead of five stages. This change, however, would be accomplished at the sacrifice of the additional information that is provided by the finer grouping that has been used.

Improper audiometric technique can be a major contributor to errors in threshold determinations, but I believe that, in the present study, errors arising from this source were insignificant. The procedure that was used to take audiograms (described earlier in the paper) is designed to elicit a maximum of accuracy in responses. Two features of this technique are important. First, by facing away from the audiometer and the tester, the subject can receive no clues from either as to when the tone is sounded. Second, the time and length of the presentation of the tone are under the control of the tester, so that he can determine, by observing the actions of the subject, whether he is truly responding to the auditory stimulus.

The importance of these errors in the present study centers about one question: In making comparisons of five-year hearing changes between two groups of employees, one exposed to occupational noise and one not exposed, will the errors in the audiogram distort the comparisons in any way that will tend to obscure whatever effect the noise may have? The answer to this question is no, if the following assumptions are valid.

(*A*) An error in threshold determination is just as likely to produce a false reversion to an earlier stage as it is to produce a false progression to a more advanced stage.

(*B*) Errors that produce false progressions to a more advanced stage are just as likely to occur in a group not exposed to noise as they are in a group that is exposed.

If these assumptions are true, and a significantly greater proportion of advances to a higher stage are found in the noise-exposed group, the differences in hearing changes may be attributed to noise, rather than to artifacts arising from audiometric errors. The assumptions would not be justified if auditory fatigue, improper audiometric technique, and testing-room noise influenced threshold determinations. As pointed out above, these potential sources of error were well under control in the plant that is being studied.

In accordance with the foregoing discussion, a change in hearing acuity during the five-year period of observation is defined as one which shows a shift to a more advanced stage, and reversions are treated as if no change had occurred.

Findings

The Effect of Noise Stress on Hearing Acuity.—Five-year changes in hearing acuity, by initial stage of high-frequency hearing loss, age at onset of observation, and over-all level of noise exposure are presented in Tables 3 through 6. Table 8 summarizes these data by showing the per cent of persons who advanced one or more stages during the five-year period. Figure 2 is a graphic presentation of the percentages in Table 8.

Table 3 and the first four rows in Table 8 describe hearing changes among persons who had a normal audiogram (Stage 0) at the beginning of the five-year study period. The data under the "Total" sections in these tables show that the proportion of persons who advanced one or more stages increased with each succeeding level of noise stress. It can be seen that among employees exposed to less than 75 db. 30.4% lost some hearing acuity. This percentage comprises 27.8% who advanced to Stage 1 and 2.6% who advanced to Stage 2. Among those exposed to 75 to 84 db. the per cent who advanced one or more stages increases to 48.1, and rises to 51.2 in the 85 to 90 db. category. It is not likely that the higher percentages

TABLE 3.—*Five-Year Change in Stage of High-Frequency Hearing Loss by Age at Onset of Observation and Over-All Level of Noise Exposure*
Initial Stage of High-Frequency Hearing Loss: 0

Stage After 5 Yr.	Age at Onset of Observation, Yr. 20–34						35–44					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	45	75.0	26	63.4	28	52.8	26	61.9	11	42.3	9	40.9
1	12	20.0	14	34.1	18	34.0	16	38.1	13	50.0	11	50.0
2	3	5.0	1	2.5	6	11.3			2	7.7	2	9.1
3					1	1.9						
4												
Total	60	100.0	41	100.0	53	100.0	42	100.0	26	100.0	22	100.0

Stage After 5 Yr.	Age at Onset of Observation, Yr. 45–59						Total					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	9	69.2	3	30.0	2	40.0	80	69.6	40	51.9	39	48.8
1	4	30.8	5	50.0	3	60.0	32	27.8	32	41.6	32	40.0
2			2	20.0			3	2.6	5	6.5	8	10.0
3											1	1.2
4												
Total	13	100.0	10	100.0	5	100.0	115	100.0	77	100.0	80	100.0

in the noise-exposed groups can be explained by chance alone.† Thus, the data seem to indicate that persons with normal hearing will suffer some loss of hearing acuity after five years of exposure to occupational noise between 75 and 90 db.

The same pattern is seen in the group of employees who were at Stage 1 at the beginning of the period (Table 4). Among

† The statistical test of significance that was used throughout this section was the chi-square test of independence with Yates' correction for continuity. The level of significance was 0.05.

TABLE 4.—*Five-Year Change in Stage of High-Frequency Hearing Loss by Age at Onset of Observation and Over-All Level of Noise Exposure*
Initial Stage of High-Frequency Hearing Loss: 1

Stage After 5 Yr.	Age at Onset of Observation, Yr. 20–34						35–44					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	8	21.6	7	15.6	5	11.4	4	11.4	5	15.6	5	12.2
1	24	64.9	30	66.7	25	56.8	22	62.8	20	62.5	24	58.5
2	5	13.5	8	17.7	13	29.5	8	22.9	6	18.8	11	26.8
3					1	2.3	1	2.9			1	2.5
4									1	3.1		
Total	37	100.0	45	100.0	44	100.0	35	100.0	32	100.0	41	100.0

Stage After 5 Yr.	Age at Onset of Observation, Yr. 45–59						Total					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	7	17.9			1	3.3	19	17.1	12	11.3	11	9.6
1	23	59.0	15	51.7	8	26.7	69	62.2	65	61.3	57	49.6
2	7	17.9	12	41.4	20	66.7	20	18.0	26	24.5	44	38.2
3	2	5.2	2	6.9	1	3.3	3	2.7	2	1.9	3	2.6
4									1	1.0		
Total	39	100.0	29	100.0	30	100.0	111	100.0	106	100.0	115	100.0

those exposed to less than 75 db. 18.0% advanced to Stage 2 and 2.7% to Stage 3, so that a total of 20.7% lost some hearing acuity. In the 75 to 84 db. and 85 to 90 db. groups, 27.4% and 40.8%, respectively, advanced to a higher stage. It should be noted, however, that the difference between 27.4% and 20.7% could easily have arisen by chance, while the difference between 40.8% and 20.7% is highly significant ($P>0.01$). Therefore, it may be inferred that when a person is at Stage 1 of high-frequency hearing loss, five years of subsequent exposure to a noise level between 75 and 84 db. may not affect his hearing, but his hearing acuity will be affected during that time period if he is exposed to a noise level between 85 and 90 db.

Among employees at Stage 2 at the beginning of the period, there is no apparent relationship between five-year hearing changes and level of noise exposure. As indicated in Tables 5 and 8, 11.9% of the employees exposed to less than 75 db. advanced to Stage 3 or 4. In the 75 to 84 db. and 85 to 90 db. groups, the respective percentages of those who advanced beyond Stage 2 are 19.4 and 10.8. Differences among these percentages are not large enough to reject the hypothesis that they come from the same population.

Even though the differences in hearing changes among employees at Initial Stage 2 can be explained by sampling variation, it is interesting to note that in each of the three age categories the proportion of persons who advanced to a higher stage is greater in the 75 to 84 db. group than it is among persons exposed to 85 to 90 db. This seems to be a paradoxical situation, in that the data suggest that exposure to the higher noise level is less damaging than exposure to the lower noise level. This may be a manifestation of an unknown bias in the data, or it may simply be the result of sampling error.

No analysis was made of the data obtained for persons at Stage 3 at the beginning of the period because the number of observations is not large enough for meaningful statistical analysis. The data are presented, however, in Table 6.

It is clear from the foregoing discussion that hearing loss during the five-year period was related to the initial stage as well as the level of noise exposure. This point may be seen more clearly in Table 7, which

TABLE 5.—*Five-Year Change in Stage of High-Frequency Hearing Loss by Age at Onset of Observation and Over-All Level of Noise Exposure*
Initial Stage of High-Frequency Hearing Loss: 2

Stage After 5 Yr.	Age at Onset of Observation, Yr. 20-34						35-44					
	Less than 75 Db.		75-84 Db.		85-90 Db.		Less than 75 Db.		75-84 Db.		85-90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0					1	2.0	1	2.4			2	4.3
1	6	18.2	5	13.9	7	14.3	1	2.4	5	13.1	2	4.3
2	24	72.7	28	77.8	38	77.6	33	80.5	27	71.1	39	83.0
3	3	9.1	3	8.3	2	4.1	4	9.8	5	13.1	3	6.3
4					1	2.0	2	4.9	1	2.7	1	2.1
Total	33	100.0	36	100.0	49	100.0	41	100.0	38	100.0	47	100.0

Stage After 5 Yr.	Age at Onset of Observation, Yr. 45-59						Total					
	Less than 75 Db.		75-84 Db.		85-90 Db.		Less than 75 Db.		75-84 Db.		85-90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0									1	0.8	3	2.2
1	3	6.9	4	7.3	2	4.7	10	8.5	14	10.8	11	7.9
2	36	81.8	35	63.6	33	76.7	93	78.8	90	69.8	110	79.1
3	5	11.3	16	29.1	7	16.3	12	10.2	24	18.6	12	8.6
4					1	2.3	2	1.7	1	0.8	3	2.2
Total	44	100.0	55	100.0	43	100.0	118	100.0	129	100.0	139	100.0

TABLE 6.—*Five-Year Change in Stage of High-Frequency Hearing Loss by Age at Onset of Observation and Over-All Level of Noise Exposure*
Initial Stage of High-Frequency Hearing Loss: 3

Stage After 5 Yr.	Age at Onset of Observation, Yr.											
	20–34						35–44					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0												
1					1	25.0						
2											1	25.0
3					3	75.0	5	100.0	5	100.0	3	75.0
4			1	100.0								
Total			1	100.0	4	100.0	5	100.0	5	100.0	4	100.0

Stage After 5 Yr.	Age at Onset of Observation, Yr.											
	45–59						Total					
	Less than 75 Db.		75–84 Db.		85–90 Db.		Less than 75 Db.		75–84 Db.		85–90 Db.	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0												
1											1	5.0
2	2	20.0	5	27.8	1	8.3	2	13.3	5	20.8	2	10.0
3	2	20.0	5	27.8	7	58.4	7	46.7	10	41.7	13	65.0
4	6	60.0	8	44.4	4	33.3	6	40.0	9	37.5	4	20.0
Total	10	100.0	18	100.0	12	100.0	15	100.0	24	100.0	20	100.0

presents the percentages of employees who lost some hearing acuity.

It can be seen that in each noise-exposure group the proportion of employees who suffered some loss in hearing acuity decreases as the initial stage increases. Thus, the more advanced a person's high-frequency hearing loss, the less likely is it that he will undergo additional hearing loss. In other words, high-frequency hearing loss appears to begin rapidly and proceed more slowly in the advanced stages.

It is also seen that the damaging effect of the noise diminishes with each successive initial stage. Among persons exposed to 75 to 84 db., the group with normal audiograms at the beginning of the period appears to have been affected by five years of exposure to the noise. The data do not conclusively indicate, however, that employees at more advanced initial stages (1 or 2) were affected. In the 85 to 90 db. group, the same phenomenon is seen, except that persons who were at Stage 1 at the beginning of the period were affected as well as those who were at Stage 0.

TABLE 7.—*Percentages of Employees Who Lost Some Hearing Acuity During the Five-Year Period*

Initial Stage	Less than 75 Db.	75–84 Db.	85–90 Db.
0	30.4	48.1 *	51.2 *
1	20.7	27.4	40.8 *
2	11.9	19.4	10.8

* A percentage accompanied by an asterisk indicates that it is not likely that chance alone can explain the difference between it and the percentage in the "less than 75 db." category of the same initial stage ($P < 0.05$).

The percentages in Table 7 are plotted on the chart in Figure 2. Free-hand regression lines for each initial stage are drawn through each set of three points. These lines are not intended to estimate with any degree of precision the function of hearing loss with respect to level of noise exposure, but are presented to clarify further the preceding discussion by illustrating graphically the relationships that have been described.

The slopes of the regression lines for Initial Stage 0 and Initial Stage 1 are about the same, and are much greater than the slope of the line representing Initial Stage 2. This illustrates the presence of an association between noise exposure and hearing loss when hearing is normal or at the first stage of high-frequency hearing loss at the

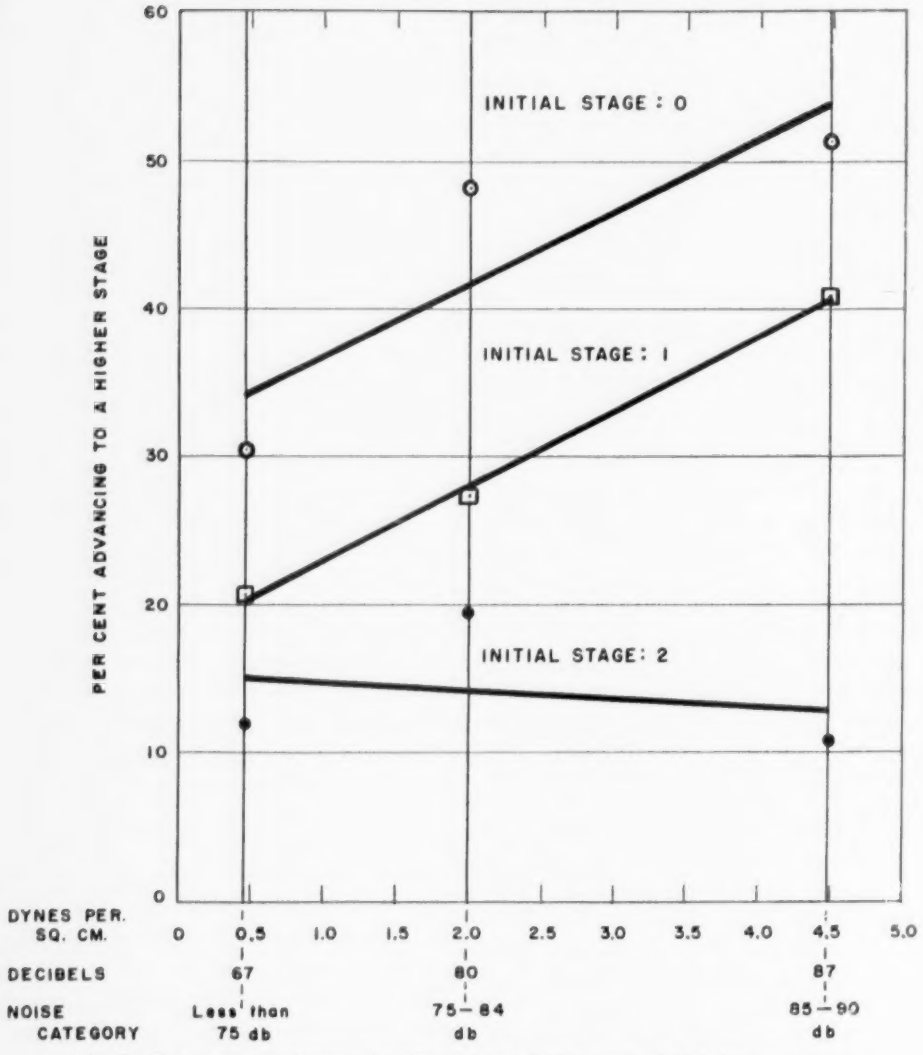

Fig. 2.—Per cent of employees who advanced to a higher stage of high-frequency hearing loss after five years of exposure to selected noise levels, by initial stage. Noise intensity is presented on an arithmetic scale, in terms of sound energy (dynes per sq. cm.).

beginning of the five-year period.‡ The small, slightly negative slope for Initial Stage 2 points up the absence of any effect of noise exposure on hearing acuity after the second stage of high-frequency hearing loss has been reached.

Another striking aspect of the chart is that each set of points and its corresponding regression line fall one below the other in the order of initial stage. This indicates that, regardless of the level of noise stress,

‡ Although the regression line for Initial Stage 1 indicates, in a general way, a relationship between hearing loss and level of noise exposure, it should be recalled that sampling error can account for the differences in rates of hearing loss between employees exposed to 75 to 84 db. and those exposed to less than 75 db. (27.4% and 20.7%, respectively).

the more advanced the initial stage the less chance there is of advancing to a subsequent stage over a five-year period.

The slowing down of the progress of high-tone hearing loss in its advanced stages, as indicated in this set of data, is consistent with what is known about the pathological changes that take place in the inner ear as a result of aging and acoustic trauma. The nerve receptors for the higher frequencies (4000 cps and above) are fewer in number than the receptors for tones in the low and middle range. This explains why loss of hearing acuity occurs initially in the high frequencies. During the advanced stages of traumatic hearing loss, when the middle and low frequencies become involved (Stage 2 and beyond), the progress of high-tone hearing loss is expected to slow down because there are more nerve receptors available in reserve to take up the functioning of those that were destroyed.

Effect of Age.—The relationship of age to hearing changes brought about by occupational noise stress is seen by examination of the data in Table 8.§ Among the three age categories in the noise-exposed groups 75 to 84 db. and 85 to 90 db., the largest proportion of employees who advanced one or more stages is consistently found among persons between 45 and 59 years of age. For example, among employees in the 45 to 59 age category at Initial Stage 1 and exposed to 75 to 84 db., 48.3% advanced to a higher stage. In the two lower age groups, this proportion was 17.7% for employees between 20 and 34, and 21.9% for employees between 35 and 44. Among persons exposed to 85 to 90 db. and at Initial Stage 1, the higher rate in the 45 to 59 age group is even more pronounced. In this category the proportion is 70.0%, as compared with 31.8% and 29.3% in the lower age groups. The same age differences are seen in persons at Initial Stage 2. It is interesting to note that this pattern is not evident among employees exposed to less than 75 db. It seems clear, therefore, that persons 45 years and over are more susceptible to traumatic hearing loss than persons below 45 years.

Extent of Occupational Hearing Loss.—These data have shown that under conditions described above persons subjected to noise stress (75 db. or more) probably undergo hearing changes that differ from those experienced by employees who do not work in a noisy environment. It should be noted, however, that among those in the noise-exposed groups who do advance to a

§ This discussion omits Initial Stage O because of the small number of persons between 45 and 59 years of this category.

TABLE 8.—*Per Cent of Employees Who Advanced One or More Stages of High-Frequency Hearing Loss After Five Years of Exposure to Selected Noise Levels, by Initial Stage and Age at Onset of Observation (from Data in Tables 3 to 5)*

Initial Stage	Age at Onset of Observation, Yr.	Per Cent Advancing to a Higher Stage After 5 Yr. of Exposure to:		
		Less than 75 Db.	75–84 Db.	85–90 Db.
0	20–34	25.0	36.6	47.2 †
0	35–44	38.1	57.7	59.1
0	45–59	*	*	*
	Total	30.4	48.1 †	51.2 ‡
1	20–34	13.5	17.7	31.8
1	35–44	25.8	21.9	29.3
1	45–59	23.1	48.3	70.0 ‡
	Total	20.7	27.4	40.8 ‡
2	20–34	9.1	8.3	6.1
2	35–44	14.7	15.8	8.4
2	45–59	11.3	29.1	18.6
	Total	11.9	19.4	10.8

* Data omitted because of small number of observations.
† Difference between percentages of noise-exposed group and control group significant at 0.05 level.
‡ Differences between percentages of noise-exposed group and control group significant at 0.01 level.

higher stage of hearing loss only a small percentage advance more than one stage over a five-year period. For example, in the "Total" section of Table 4 (Initial Stage 1), it is seen that 24.5% of those exposed to 75 to 84 db. advanced one stage, but only 1.9% advanced two stages, and 1.0% three stages. In the 85 to 90 db. group, 38.2% advanced one stage and only 2.6% two stages. The same pattern occurs in the data presented in Tables 3 and 5.

Comment

There are a wide variety of opinions regarding maximum safe noise levels. Estimates, in terms of the over-all level only, vary from about 80 db. (Goldner[12]) to 115 db. (Davis[13]). Tentative standards, specified by noise spectra, have also been proposed. Hardy's[14] criterion for prolonged exposure to steady-state noise is 100 sones, which has a peak of 115 db. in the octave band 37.5-75 cps and a minimum intensity of 91 db. at 2400-4800 cps. A criterion cited by Parrack[15] is similar to Hardy's.

The findings of this investigation raise the following points concerning the establishment of safety criteria:

1. It is generally agreed that a noisy safety criterion should be described in terms of sound pressure levels for each octave band to take into account the varying effects of different frequencies in the noise spectrum. The data presented in this study do not provide the basis for such a criterion, but they do suggest that a standard for steady-state noise, regardless of its spectrum, would have to be one whose over-all sound pressure level is less than 90 db.

2. There is evidence in the data that some employees in the 75 to 84 db. category whose audiograms were normal at the beginning of the study period did suffer some loss of hearing acuity as a result of five years of exposure to the noise. For most of these men, however, the loss did not go beyond Stage 1. This brings up an important question. Since a moderate high-frequency hearing loss, as defined here, probably does not incapacitate a person in any way, socially or in his pursuit of a livelihood, should a noise which produces damage to this extent be considered unsafe? To answer this question, a safety criterion must also include a definition of "unsafe" in terms of the kind and extent of hearing loss the noise produces.

3. Employees in the 75 to 84 db. category at Stage 1 or 2 at the beginning of the study period did not appear to be affected by the noise after five years of exposure. It is not known at the present time whether the cumulative effect of this noise over a longer period of time will produce some additional loss in hearing acuity. Therefore, it is believed that a noisy safety criterion must specify the number of years of exposure that will elapse without any loss in hearing acuity that may be ascribed to the noise.

4. The data in this study have shown that the effect of noise varies with age and degree of hearing loss, so that these factors must be considered in developing safety standards.

5. Considerable variation among persons in their susceptibility to traumatic hearing loss is seen in this study and has been noted by several investigators.[5,10,16-18] Hence, it is possible that a noise level may be hazardous for only a small portion of the exposed personnel. To reduce the noise level to a point where no one will be affected may be uneconomical or even impossible in instances where the irreducible minimum in noise reduction has been achieved. Under these circumstances it becomes necessary to assume a certain risk and define safety criteria on the basis of the proportion of employees one expects to be protected. An example of this approach is seen in the 5% deafness risk criterion cited by Parrack.[15]

Many studies have been undertaken to examine the effect of occupational noise on hearing acuity, but there remains a great deal of disagreement on the subject. One reason for the diversity of conclusions is the scarcity of adequate data, such as valid and

reliable audiograms, proper noise measurements, and long-term follow-up information. Another reason, however, is the lack of uniformity in methods of study. Important elements of methodology include the formulation of specific objectives, definitions of basic measurements (noise levels and hearing loss), sampling procedures, the design of the investigation, and analytical techniques. The efficiency of study methods is as essential to the validity of the conclusions as the accuracy of the measurements. If these methods could be standardized, greater consistency among various studies may be attained.

Summary and Conclusions

The audiograms of 1049 male employees of a manufacturing plant have been utilized to study the relationship of occupational noise exposure to five-year audiometric changes, taking into account two additional variables: age and degree of hearing acuity at the onset of the study period.

Although it is preferable to specify noise levels by octave-band analyses, the large variety of noise sources in the plant and the frequent movement of employees throughout the working day made this kind of noise measurement unusuable for the present study. Accordingly, each employee was placed into one of three over-all noise level categories: less than 75 db., 75 to 84 db., and 85 to 90 db. Most of the noise in the plant is continuous and of the steady-state type. Employees exposed to impact noise were excluded.

Measurement of hearing acuity was based on the pattern of the audiogram. Five stages of high-tone hearing impairment were defined, Stage 0 representing normal hearing and Stages 1 through 4, progressive stages of neural deafness. Hearing loss during the five-year period was specified as the difference between the hearing acuity of the worse ear at the beginning of the period and the hearing acuity of the same ear five years later.

Statistical analyses of the data support the following conclusions:

1. The hearing acuity of persons with normal audiograms (Stage 0) may be affected by five years of subsequent exposure to steady-state occupational noise ranging from 75 to 90 db.

2. The hearing acuity of persons with Stage 1 high-tone hearing loss may be affected by five years of subsequent exposure to steady-state occupational noise of 85 to 90 db. but not by noise between 75 and 84 db.

3. The hearing acuity of persons with Stage 2 high-tone hearing loss is not affected by five years of subsequent exposure to steady-state occupational noise up to 90 db.

4. Among employees exposed to noise stress who do advance to a higher stage over a five-year period, only a small percentage advance more than one stage.

5. High-frequency hearing loss begins rapidly and proceeds more slowly in its advanced stages.

6. The damaging effect of noise stress diminishes with each successive initial stage.

7. Persons 45 years and over are more susceptible to traumatic hearing loss than persons below 45 years.

The data have also pointed up some of the complexities involved in developing safety standards. In addition to describing the noise by its spectrum, type, and temporal character, this study demonstrates that noise safety criteria must also take into consideration the number of years of exposure, the kind of hearing loss the noise produces, the age and degree of neural deafness of the exposed personnel, and variation among persons in their susceptibility to traumatic hearing loss.

Dr. Antonio Ciocco and Dr. Donovan G. Thompson, of the Graduate School of Public Health, University of Pittsburgh, aided in this study.

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Recruitment Testing

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A basic requirement for a new test designed, like the loudness balance test, to separate various clinical groups of deafness is that there must be a significant statistical difference between the mean values for the different categories. If the figures characteristic of the two principal groups, recruiting and nonrecruiting deafness, fall within one standard deviation, there is no justification for giving the test any diagnostic value. And although it ought to be clear that each proposed test should have had a thorough comparison with a direct loudness balance test, this has been omitted oftener than included.

Difference Limen Tests

In the recent past much has been written about the diagnostic importance of the intensity difference limen (IDL). This test is based on two assumptions, viz., that the number of the just-noticeable differences is the same in a recruiting ear as in a normal ear and, consequently, because the loudness in a recruiting ear increases more rapidly than normal, the just-noticeable differences are smaller than normal. However, neither of these assumptions need be true. Indeed, one would intuitively think that in an ear where the number of functioning hair cells is greatly reduced, or the cells are of a lower functioning capacity, the intensity discrimination of the ear would be somewhat poorer than that of a normal ear with its unimpaired structures.

Together with Hirsh and Goodman[1] I was able to prove that, if the IDL is measured by comparing two different tones, the values obtained in the different clinical categories are indistinguishable from one another (Fig. 1). Another method with modulated tone, originally suggested by Lüscher and Zwislocki,[2] has been claimed to accomplish the clinical separation inside an intensity modulation of 2%-16%. These figures correspond to 0.18 to 1.5 db., the range of 0.18 to 0.54 db. signifying recruiting deafness. It is obvious that the application of even the simplest statistical measures to these figures in ordinary clinical groups will result in considerable overlapping.

This was demonstrated by Fournier,[3] who showed in 50 normal ears that at the higher frequencies even a difference limen as small as 0.4 db. is inside one standard deviation of the normal mean (Fig. 2), and by Lund-Iversen,[4] whose results indicated that the just-detected modulation change had similar mean and range values in all clinical groups.

The idea that intensity discrimination is better than normal in a recruiting ear suggests that the ear's capacity to carry out a normal pitch analysis is impaired. This as-

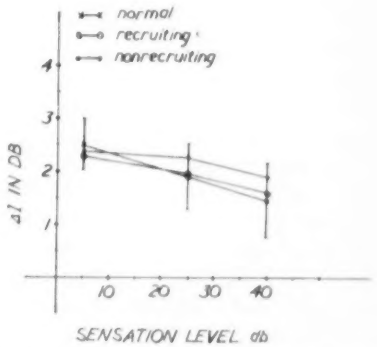

Fig. 1.—The size of the IDL in normals and in cases with recruiting and nonrecruiting deafness. The measurements were made with two separate tones. The vertical lines indicate the area of one standard deviation around the normal mean. Data from Hirsh, Palva, and Goodman.[1]

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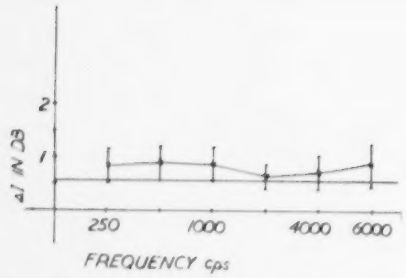

Fig. 2.—Normal values for IDL at various frequencies; data from Fournier.[5] The measurements were made with a modulated tone. The vertical lines indicate the area of one standard deviation around the normal mean. The 0.54 db. straight line, defined as the upper limit in complete recruitment by Luscher and Zwislocki,[2] is inside one standard deviation at all frequencies.

sumption is clearly on a sounder basis than the ideas underlying the IDL tests, because the number of just-noticeable frequency steps on a certain frequency range ought to be standard regardless of the degree of hearing loss.[5] Furthermore, we know from clinical experience that perceptively deaf ears often present the phenomenon of diplacusis, which means that the pitch of a tone as heard by the hard of hearing ear differs from that heard by the other (normal) ear.

The clinical use was proposed by Schubert,[6] who noted that the frequency difference limens (FDL) were one and one-half to four times as large in ears with recruiting deafness as in a normal ear. This finding was strongly contradicted by Azzi's[7] large series of 241 cases demonstrating that the FDL's were generally in normal limits in cases of recruiting deafness but considerably increased in cases of retrocochlear nerve pathology. O. Meurman's[8] enthusiasm for the value of the FDL test in the detection of recruitment is not supported by his results. Although the standard deviations were not calculated, the means and range of observations clearly indicate that there is considerable overlapping of results in the different clinical categories.

Fournier[3] made careful studies of the FDL, using also statistical measures to determine the normal variability of the individual observations. His results showed that generally the FDL's were clearly increased in perceptive deafness, but there were both recruiting and nonrecruiting cases in which the FDL was normal. In Fournier's opinion, the FDL figures can be more accurately correlated with the type of recruitment function and with etiology than just with simple positive recruitment.

It seems that measurement of the FDL in the present state is not yet applicable in clinical use. In my experience, the results vary individually within wide limits, and repeated measurements of FDL on one person do not always give consistent values. The type of modulation employed may affect the results considerably. One also wonders why the FDL should always be increased in recruiting deafness considering that the impaired ear may perceive a tone of lower and of higher pitch than the normal ear. A factor of uncertainty is also introduced by the fact that the tone in a defective ear may lose its tonal character and sound more like a buzz, or it may present a complex mixture of tones instead of just one single tone perceived by the normal ear.[5]

Masking Tests

On the basis of Wegel and Lane's[9] masking curves of pure tones by pure tones, de Bruine-Altes and Huizing[10] suggested this type of test for detection of recruitment. In this monaural test the lower one of a pair of tones was used as a masking stimulus, and, in the authors' opinion, it was necessary for one tone to have a normal or nearly normal threshold. Measured relative to the sensation level, nonrecruiting deaf subjects were found to show a normal masking pattern, whereas in recruiting deafness there was either diminished or increased masking, depending on the thresholds of the pair of tones.

Instead of relating masking to sensation level, it is more logical to relate it to the masking pattern in a normally hearing subject, as done by Tumarkin.[11] He concluded that the test separates conductively deaf

persons from all others, since in conduction deafness the sound intensity reaching the inner ear is reduced and masking is considerably less than that in a normal ear. In perceptive deafness the intensity at the inner ear is not reduced and therefore the masking is identical to the normal.

De Bruine-Altes and Huizing's hypothesis maintains that it is not the intensity level at the inner ear but the loudness level that is decisive in producing the deviating pattern in recruiting deafness. This is somewhat difficult to understand, as the loudness in a recruiting ear only seldom exceeds that in a normal ear, and the masking therefore should be comparable to normal.

Further data on this question were presented by de Maré and Rösler,[12] who used an automatic recording of the masked threshold by means of a self-recording audiometer. In general, they observed that conductive deafness deviates from other types of hearing impairment by less than normal masking. However, in their experience, many recruiting perceptively deaf ears showed abnormally large masking.

The great drawback in pure-tone masking tests is the interaction of the masking and masked stimuli, which renders the exact determination of the masked threshold difficult. I have made measurements on 12 normal ears with a 1000 cps masking tone

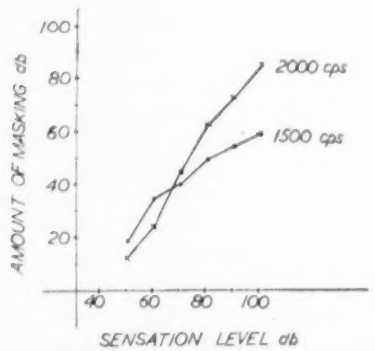

Fig. 3.—Masking effect of 1000 cps pure tone upon 1500 and 2000 cps tones in 12 normally hearing subjects as a function of sensation level. For the 2000 cps tone the masked thresholds become considerably larger than those of a 1500 cps tone as the sensation level increases.

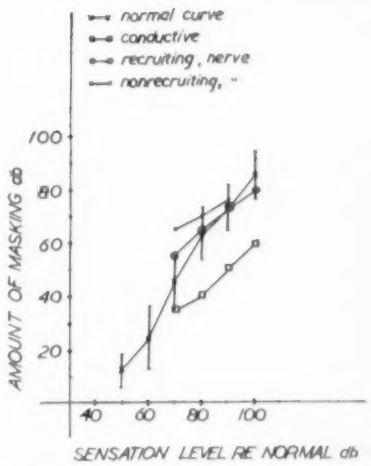

Fig. 4.—Masking effect of 1000 cps pure tone upon 2000 cps tone in various groups of deafness. The vertical lines indicate the area of one standard deviation for the normal 2000 cps curve. It appears that only the conductive group differs significantly from the others.

at 50 to 100 db. sensation levels re normal threshold using 1500 and 2000 cps as masked tones. The curves in Figure 3 show that at lower sensation levels masking is greater for the 1500 cps tone but after 70 db. this curve levels off, while the 2000 cps curve continues its course and shows at higher levels considerably more masking than the 1500 cps curve.

It is obvious that only the 2000 cps curve has enough steepness to be used in differential diagnosis. The results relative to this curve in 13 conductively deaf ears, 27 ears with recruiting perceptive deafness, and in 10 ears of nonrecruiting perceptive deafness are presented in Figure 4. The thresholds were determined at 70 to 100 db. levels relative to normal threshold with a 1000 cps masking tone and 2000 cps masked tone. It appears that only the conductively deaf group differs significantly from all others, showing clearly less masking than normal. The perceptively deaf ears are closely comparable to the normal; the major deviations at the 70 db. level are partly produced by the initially great threshold loss.

The pure-tone masking results presented seem to substantiate Tumarkin's [11] claim that

this test is useful in separating conductive deafness from all other types, but it does not support studies suggesting a greater differential diagnostic importance of this test. Furthermore, in this study the standard deviations were too large even in a normal group (from 6 to 12) to assign differential diagnostic value to any small deviations from the normal.

Langenbeck,[13] of Germany, devised another type of masking measurement. He employed a white-noise masking stimulus and measured the auditory thresholds in the presence of this noise. He concluded that in the retrocochlear nerve type of deafness the masking produced by white noise was greater than in the other groups, which showed a masking predictable on the basis of the critical band width.[14] However, control experiments [15] with accurately measured stimuli failed to show any differences between recruiting and nonrecruiting perceptive deafness, and the masking produced was in all cases consistent with that calculated on the basis of the critical band width.

At the measurement of pure-tone thresholds in interrupted noise at a rate of 1 to 12 interruptions per second there was no significant difference between the various clinical groups of deafness.[16] Speech intelligibility in a 95 db. continuous white noise with a S/N ratio of +10 was very poor in some perceptively deaf ears, whereas normal subjects and conductively deaf persons reached high articulation scores. However, low intelligibility occurred in perceptive deafness regardless of the presence of recruitment.[16]

Auditory Fatigue and Adaptation Tests

The same reasoning as employed in most of the masking tests has also been used in the measurement of auditory fatigue for recruitment. In conductive deafness fatigue will be less than normal owing to diminished sound intensity in the inner ear, whereas in perceptive deafness fatigue will be normal or even greater because the effective level of the stimulus in the inner ear is not reduced. In recruiting deafness especially, the fatigue produced should be greater than normal, because as heard by the recruiting ear the fatiguing tone is louder. Using this reasoning, Huizing [17] proposed a special test to separate recruiting cases from the other cases: During three minutes' stimulation at 2000 cps and a 30 db. sensation level the resulting fatigue is in recruiting deafness between 19 and 22 db., whereas in all other groups it varies between 5 and 15 db.

Huizing measured the fatigue "immediately" after cessation of the stimulus. I [18] made a two-minute continuous record of the poststimulatory recovery of the ear in this test with a self-recording audiometer. The results showed that in normal persons and in those with conduction deafness the recovery was similar but subject to considerable individual variation. Some perceptively deaf ears also showed good recovery, but in others there was clearly an abnormally large poststimulatory fatigue. However, this abnormality was not associated with recruiting deafness alone but was found also in nonrecruiting perceptive deafness.

Perstimulatory fatigue, or adaptation of the ear, has also been used as a differential diagnostic sign. Hood [19] observed that the change in the loudness of a continuous stimulus is abnormally large in recruiting perceptive deafness and that some of these ears rapidly fail in performance during sustained stimulation. This finding, which is easy to confirm, was not frequent in my experience,[20] occurring in less than 20% of the cases with recruiting deafness. Therefore it does not seem possible to identify this test with a direct loudness balance test although very large losses seemed to occur only in recruiting deafness.

A special perstimulatory adaptation test is continuous threshold recording. It was suggested by Hood [21] that a large threshold sensitivity loss during continuous recording is an indication of end-organ deafness. Kos [22] recently supported the opposite view,

viz., that abnormal sensitivity loss is associated with nonrecruiting perceptive deafness, especially in patients with acoustic tumors.

A series of 39 ears have been tested by me[23] at 56 frequencies for four-minute threshold tracing. The results showed, like those obtained in high-intensity perstimulatory adaptation tests, that an abnormal loss in sensitivity is not common enough to give reliable clues to differential diagnosis and can be associated with both subgroups of perceptive deafness. This view was also expressed earlier by Dix and Hood[24] and by Reger and Kos.[25]

Width of the Automatic Threshold Tracing

The amplitude of the threshold tracing in self-recording audiometry was also recently correlated with loudness balance tests in our laboratory.[26] It appeared that the size of the threshold tracing was closely correlated both with the type of recruitment function and with the tested frequency. In asymptotic recruitment, tracings generally were clearly less than 5 db., while all cases with delayed recruitment and many of those with straight-line recruitment showed tracings comparable to normal. It was observed that at 2000 cps and at the higher frequencies there was a good opportunity to associate recruiting deafness with small tracings, but at lower frequencies, especially at 125 and 250, the threshold tracings were generally comparable to normal in spite of complete recruitment. Also, there were some cases showing excursions characteristic of recruitment although they had normal hearing or belonged to other nonrecruiting categories.

Speech Tests

Poor intelligibility for speech has also been regarded as a phenomenon paralleling recruiting perceptive deafness. The articulation score generally decreases with increasing threshold loss, and in severe recruiting cases intelligibility is very poor. There is general agreement upon these points among several investigators.[1,27-29] Recently Schuknecht and Woellner,[30] and Walsh and Goodman[31] reported, in dealing with acoustic tumors, that intelligibility for speech was very poor in these nonrecruiting cases. This is contradicted by an earlier study of Eby and Williams[32] in which cases with acoustic tumors generally showed moderately good discrimination, about 60%-70%. As the masking used in all these studies appears to have been quite correct, further data are required for a clear opinion on this question.

From the many tests reviewed above, two emerge at present which may be regarded as closely associated with the presence of recruitment. We use the self-recording audiometer to reveal the growth of loudness in the form of small threshold excursions in cases where one cannot obtain a detailed recruitment function with the balance tests. This test is always made at a fixed frequency during some minutes' stimulation, and preferably in the range 2000 to 4000 cps. Another test, which is made in most cases of perceptive deafness, and in all cases before an operative procedure, is the speech audiogram. In my experience,[1,29] poor intelligibility shows good agreement with direct loudness balance tests providing the hearing loss is moderate or severe.

Other indirect tests are largely used in the experimental sense, as it is felt that they do not yield enough information regarding the presence of recruitment. However, their use may sometimes be also indicated to make a distinction between the two main groups, perceptive and conductive deafness. This is particularly the case if the middle-ear pathology is such that it limits the movements of the windows and results in lowered bone-conduction values also in conductive deafness.[33]

Summary

Various indirect recruitment tests are reviewed and evaluated, i. e., tests for intensity and frequency difference limen, masking, auditory fatigue and adaptation, self-recording threshold audiometry, and speech

intelligibility. It is concluded that recruitment should be measured with direct tests in all cases in which this is technically possible. In cases with nearly the same threshold loss in both ears and at all frequencies, self-recording threshold audiometry and speech discrimination tests were found most reliable for predicting the presence of recruitment.

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Action of Proteolytic Enzymes on Cooked Meat

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The use of proteolytic enzymes to digest cooked meat impacted in the esophagus is a distinct aid in treatment of such a condition. Crystalline trypsin (Tryptar) was used advantageously in a case reported previously.[1] The results of a series of experiments to determine the comparative rate of digestion of cooked meat by trysin, Caroid (papain), and proteinase A, with isotonic saline as a control, were also noted. Caroid produced the best result in vitro. The Caroid was not sterile, however, for a Gram-positive micrococcus was isolated from it by culture. For in vivo use, crystalline trypsin was recommended since it was sterile, the solution could be made easily, and the mixture could be given readily.

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Papain is the dried, powdered, and whole latex of the green fruit of Papaya carica.[2-4] Caroid is one of the commercial names for papain. Two highly purified enzymes, crystalline papain and crystalline chymopapain, have been isolated from the latex of P. carica. It was of interest to determine the degree of digestion of cooked meat produced by these and other enzymes.

Nine cubes of cooked meat measuring about 0.5 cm. on a side were put into test tubes. The details of the experiments are shown in Table 1. The pH of the solutions were determined once with nitrazine paper. The digestion was done at 37.5 C for 24 hours. All of the tests were done twice. In all instances, a fine residue was present when maximal digestion was obtained.

The digestion of cooked beef was accomplished by all of the proteolytic enzymes (Table 2).

TABLE 1.—*Digestant Solutions*

Agents and Amounts of Agents Used	Solvent	pH of Solution (Approximation)
Caroid,* 0.5 Gm.	5 cc. of isotonic saline	5.0
Crystalline papain,† 0.125 Gm.	5 cc. of isotonic saline	5.0
Crystalline chymopapain, 0.125 Gm.	5 cc. of isotonic saline	6.0
Tryptar,‡ 0.125 Gm.	5 cc. of Sorenson's phosphate buffer solution	7.0
Pepsin,‡ 0.125 Gm.	5 cc. of isotonic saline	4.5
Crystalline papain, 0.125 Gm., and crystalline chymopapain, 0.125 Gm.	5 cc. of isotonic saline	6.0
Crystalline papain, 0.125 Gm., and crystalline trypsin, 0.125 Gm.	2.5 cc. of isotonic saline and 2.5 cc. of Sorenson's phosphate buffer solution	6.25
Crystalline papain, 0.125 Gm., and pepsin, 0.125 Gm.	5 cc. of isotonic saline	4.5
Crystalline chymopapain, 0.125 Gm., and crystalline trypsin, 0.125 Gm.	2.5 cc. of isotonic saline and 2.5 cc. of Sorenson's phosphate buffer solution	7.0
Crystalline chymopapain, 0.125 Gm. and pepsin, 0.125 Gm.	5 cc. of isotonic saline	4.75
Isotonic saline	5 cc. of isotonic saline	5.0

* The caroid was supplied by the American Ferment Company, Inc., New York.
† The crystalline papain and the crystalline chymopapain were supplied by Winthrop-Stearns, Inc., New York City.
‡ The tryptar and the pepsin were supplied by The Armour Laboratories, Chicago.

TABLE 2.—*Percentage of Digestion*

Agents Used	Time in Hours							
	1	2	3	4	5	6	12	24
Caroid	30	35	40	50	60	70	90	90
Crystalline papain	15	25	30	40	45	55	70	75
Crystalline chymopapain	10	15	20	25	30	35	40	50
Crystalline trypsin	5	10	20	30	40	45	60	75
Pepsin	0	0	0	0	0	5	10	20
Crystalline papain and crystalline chymopapain	15	20	25	30	40	45	55	70
Crystalline papain and crystalline trypsin	20	25	30	40	50	60	80	80
Crystalline papain and pepsin	5	10	15	20	25	30	40	50
Crystalline chymopapain and crystalline trypsin	15	20	25	30	40	45	60	75
Crystalline chymopapain and pepsin	15	20	25	30	40	45	60	75
Isotonic saline	0	0	0	0	0	0	0	0

Caroid was the most effective. Caroid, however, was not sterile. Crystalline papain produced a high degree of digestion during the first four hours.

Crystalline papain[3] is sterile and is active over a pH range of 2.5 to 12. The use of crystalline papain is recommended in an esophagus which contains impacted cooked meat.

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Progress in Otolaryngology

Functional Examination of Hearing

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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It is our purpose in these abstracts to present the subject matter in such a form that it will be useful to the practicing otologist whose office is not equipped with the elaborate armamentarium required by the scientific audiologist, whose field is highly specialized but more limited in scope.

Ewertsen [1] reports the program employed in Denmark for evaluating hearing aids and prescribing them. Three hearing centers have been established to take care of the entire Danish area with its four million inhabitants. The individual hearing aids are controlled by an "artificial ear" at the hearing center. The frequency curves are plotted and filed so as to have a detailed account of the commonest defect. The fitting of the hearing aid is based on a close cooperation of the engineers and physicians. Hearing aids of inferior quality have been weeded out by means of the artificial ear. The main object of the program is to provide the patient with a reliable hearing aid with satisfactory amplification and a good after-care educational program, aiming at a safe restitution of the patient after a probably difficult period of adaptation. A reexamination of the first 800 patients showed that only 4% failed to use their hearing aid. The total expense for each person is approximately $7.00 plus the cost of the hearing aid.

In a survey of 1459 patients, Kodicek and Garrad [2] reported that 90% found the hearing aid useful. The conductive-deafness group derived more assistance from the aids than did the perceptive-deafness group. The more severely deaf were more appreciative. With advancing age the aids were used for shorter periods and less often. One-half used the aids at work. Considerable loneliness appeared among the elderly deaf. The main criticisms were "the aids were cumbersome" and the "leads broke too often." For the majority the Medresco proved satisfactory. (Hearing aids are government-furnished in England.)

Watson [3] describes the plan and program employed in the use of hearing aids for severely deaf children. Very early testing and screening of young children is carried out with the aid and help of the Maternity and Child Welfare Service. When a case is found to have some useful residual hearing, an aid is recommended, to be used for variable periods of time until the child is willing to wear it continuously. In some children this may be as early as 18 months of age. With preschool children no formal training is attempted. The aim is that the child learn to comprehend speech. Thus listening to and watching for speech will take place at all times and will enrich the child's growing understanding of both language and his environment. This informal use of the aid is carried out at the infant and nursery ages, namely, 3 to 7 years. For children of 7 and upward group hearing aids should be provided for the classroom for more formal training. Satisfactory equipment, proper environment, and—above all—adequately trained teachers are essential

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to make an effective program for teaching these children.

Wallace et al.[4] made a survey of the facilities available in New York City which provide services for patients with hearing problems. Twenty-four agencies were studied. Fourteen were located in general hospitals, six in specialty hospitals, and four in detached services. Some of these agencies were rather limited as to the facilities available or specialized as to the type of patient served. This group feels that in order for a hearing center to be considered adequate it should provide complete services for the diagnosis, treatment, and rehabilitation of children and adults who have a hearing impairment. It should care for patients referred to it from all sources, such as the practicing physicians, commmunity agencies, school health and child health services, and industry. It should provide interpretation and guidance to the patient and his family, and should assist the patient and his family in making plans for total rehabilitation. It should also be a training center for professional personnel of all types and should perform research. The physical facilities should include sound-treated areas for diagnosis and therapy, reception and observation rooms, ear-mold and electronics laboratories, and other areas, adding up to 2500 sq. ft. of floor space. The personnel should include medical personnel (as the otologist and pediatrician) and paramedical personnel (as the audiologist, speech therapist, nurse, social worker, and psychologist). They recommend also that the hearing centers be developed in hospitals, especially large general hospitals connected with a medical school. A program of this nature is very costly, and the general community must be prepared to assist in financing it.

Henner et al.[5] describe the team approach to hearing and speech disorders as carried out at the Michael Reese Hospital in Chicago. Impairment of communication is far-reaching in its effects on a patient. The causes for the condition may be obscure as well as multiple. To make a proper diagnosis and to help such people, the procedure employed should include an initial pediatric or medical examination, otological survey, audiological and speech examinations, social service studies, and occasional special psychometric, psychiatric, neurologic, orthopedic, and ophthalmologic examinations. The otologist, clinical audiologist, and speech pathologist cooperate to decide what type of therapy, hearing aid, and special training is needed and how these things can be carried out with respect to the patient's education. The medical social worker secures information essential to diagnosis and treatment and maintains contact with helpful social agencies. The clinical psychiatrist helps in solving diagnostic problems and, if necessary, aids in overcoming resistance to rehabilitation. With this team approach it is possible to pursue the resolution of the difficulty from a multiphasic point of view to accomplish a common goal, namely, the rehabilitation of the child or adult who has an impairment in communication.

Getz[6] says that the curve in hereditary deafness is nontypical and there is no prognostic value in auditory training.

In the recommendation of a hearing aid, parents should be reminded that a child's educational future depends on the visual presentation, with the auditory avenue serving as an auxiliary tool, in the development of speech and lipreading skills.

Yenrick[7] discusses trends in education of the deaf. He stresses the importance of early diagnosis, and formal education of the very young in the home. He also points out that the incidence of total deafness in children is rare. In an evaluation of pure-tone audiometrics done in the Pennsylvania School for the Deaf, of 433 subjects ranging from 4 to 20 years of age, only 2 gave a clinical indication of having total and complete loss of hearing for all frequencies from 125 through 8000 cps. Functionally speaking, the majority of the students were profoundly deaf without the use of amplification and proper teaching techniques, but clinically they were not totally deaf. Educa-

tional placement of the child must depend on available local facilities, the individual needs of the child, and his physical growth and language progress made throughout his early formative years. The educational needs of the auricularly handicapped child rest mainly with the parents and the attending physician. The child's complete future rests upon the decision made by these persons. An appended index to the article gives a list of several nonprofit organizations which offer a wealth of excellent information for the lay and the professional person.

The John Tracy Clinic[8] carried on a study to evaluate the success of the correspondence course distributed by the Clinic and to collect information on the kind and amount of educational advice given to parents of young deaf children. The information came from the first five thousand families enrolled in the course. At the time of application 854 of the children had not yet had an evaluation of the extent of their hearing loss; 1504 had been classified as hard of hearing, with average losses up to 75 db. through the speech range, and 2645 had been classified as severely or profoundly deaf, with losses greater than 70 db. Ninety-two per cent of the parents in this study had no previous knowledge of deafness and the education of the deaf; seventy-one per cent of this group received advice from the physician concerning their deaf child's education; seventy-seven per cent of the physicians discussed the teaching of speech with the family. Only 715 of the families reported they had received specialist treatment at the time of the application for the course. This evaluation of medical recognition of educational problems of deafness reflects knowledge on the part of the entire profession and not specifically on the part of otologists. Seventy-one per cent of the physicians knew the names of the schools for the deaf nearest to where the families lived and referred them to other organizations offering help. These figures indicate that physicians have been doing an excellent job with a difficult but essential educational problem.

Pfeiffer[9] reports on the mechanics of the findings and the results of a hearing survey conducted in a school unit in which the total population of the unit area is less than 30,000. The program embraced full cooperation of the school health department, local otolaryngologists, and the parents of the children. In cases of financial hardship the school nurses made a social-service investigation. The follow-up for drawing of conclusions was done by home social-service visits by the school nurses. It was found in retrospect that many abnormal hearing problems became normal, especially in the classification of hypertrophied lymphoid tissue due to these factors: treatment of allergic rhinitis, recession of lymphoid tissue at puberty, disappearance of acute infections, and examinations made during the fall of the year giving a finding differing from that revealed in examinations made during the spring, due to changes in the upper respiratory tract.

Kodman Jr.[10] compared audiometric findings with teacher identification of hearing loss to determine the accuracy of the latter method. A group of 665 elementary school children were investigated. He found that 3% of the hearing-loss cases were correctly identified by both methods. Five per cent referred for audiometric examination were found to have normal hearing. Sixteen per cent of hearing-loss cases were missed by the teachers. Seventy-six per cent were correctly identified by both methods as being normal. Although teacher identification of hearing losses is better than chance, it is recommended that audiometric examination be relied upon to detect hearing losses in school children.

Harris et al.[11] propose a new formula for predicting speech hearing loss from the audiogram. They discuss previous systems used, namely, average pure-tone loss, Sabine's, Fowler's, and Fletcher's systems. The new system is based upon the statistic of multiple regression prediction. The partial regression coefficients for each frequency are used as weights in the final multiple regression prediction equation. Hearing

losses at each relevant frequency are multiplied by their respective weights, added, and the sum is the prediction of the speech hearing losses for those persons comprising the whole group of patients from whom the original data were derived. All the systems were tried out on a series of 197 partially defective ears with fairly complete diagnostic information available. They found that no one predictive system is superior for all audiograms, since some audiometric patterns can always be found to exploit the strength or weakness of any one system. They found that the most stable system for predicting speech hearing loss was an average of the multiple regression equation prediction and the three-average prediction. (The average of these two systems was recommended to be used in the Navy to assess speech hearing loss.) To make the computation easy a cumulative table has been prepared and is presented at the end of the article. The entries in the table of the hearing losses for the five frequencies 500, 1000, 2000, 4000, 6000 cps are added, and the sum is the predicted speech loss.

Goldstein [12] tested 20 persons suspected of having nonorganic hearing loss by conditioning electrodermal responses (EDR) to tone and also by conventional audiometric procedures in an attempt to establish a reliable pure-tone audiogram. He found that the EDR test as it is currently used in cases of nonorganic hearing loss can provide information in many patients which other audiometric procedures fail to produce. However, this EDR test fails sufficiently often that the lack of electrodermal responses to tone cannot always be equated with an organic hearing loss.

Bordley [13] gives a thorough evaluation of the psychogalvanic skin resistance (PGSR) test. He feels that PGSR audiometry is a valuable adjunct to other methods for hearing evaluation. It is particularly useful in studying the hearing of young children and certain adults. Children under 6 years of age cooperate poorly, so that only two methods of pure-tone audiometry are at all reliable, namely, play audiometry and PGSR audiometry. Play audiometry, where there is good cooperation and adequate communication, offers certain definite advantages. It tests the whole hearing mechanism. However, where hearing determinations are necessary in the brain-injured child who cannot cooperate, such as in cerebral palsy, in the child whose hearing loss has delayed his ability to communicate, or in the child under 3 years of age, PGSR audiometry offers a logical solution. In all types of hearing determinations in young children retest is required to establish a stabilized threshold. In evaluating hearing in young children one should keep in mind the difference between apparent and potential thresholds. The PGSR test can be done with relatively little difficulty in children 18 months of age who have no severe brain damage. Failure increases greatly in testing children under 18 months of age.

The chief difficulties encountered in testing the hearing of children by PGSR are found, first, in establishing conditioning in certain brain injuries; secondly, in the building of a team capable of handling the test situation, and, finally, in obtaining an unbiased interpretation of the skin resistance tracings.

PGSR audiometry is useful in testing certain adults. Reports of various clinics which have used the test in cases of psychosis, and malingering warrant its continued use. This test measures reception rather than perception of sound. It offers a clinically practical approach for objective determination of thresholds of hearing for pure tones. It is an accurate test. In a series of 20 persons with impaired hearing, of whom 30 ears were tested, an average difference of less than 5 db. was found between subjective and objective audiometry throughout the sound spectrum. This test can be employed in the animal experimental laboratory. It does not necessarily evaluate hearing at the cortical level. Its accuracy is greatly enhanced when undertaken by highly trained personnel. The PGSR test is not

yet free from failure and probably never will be.

Haskins [14] reports on the value of psychogalvanic skin resistance audiometry in differentiating and diagnosing hearing conditions in adult patients. This procedure tests the function of the end-organ and provides a measurement which assists in differentiating between the receptive hearing function and the combined function of receptive and perceptive hearing mechanisms. This information is invaluable in planning a feasible course of therapy.

Maspétiol, Pigement, and Tronche [15] discuss the psychogalvanic reflex. This is produced in two ways: in a nonconditioned subject at a level of 50-60 db. above threshold as an effect of surprise and in a conditioned subject as an expression of his auditory threshold. At the uncomfortable loudness level it occurs regularly, even when surprise is excluded. The psychogalvanic reflex gives useful information about the hearing in children where subjective audiometry is impossible. A lowered uncomfortable loudness level with a psychogalvanic reflex at less than 50-60 db. is indicative of recruitment and cochlear injury; in meningitis this test for uncomfortable loudness level may help in distinguishing between the reversible cochlear and nonreversible nerve deafness. In general, disagreement between results of subjective and objective audiometry (with exclusion of functional cases) points toward central origin as the cause of the hearing impairment.

Poch-Vinals and Mitjardu [16] made a comparative study of the loudness-balance and difference-limen tests in 29 cases of various types of deafness. They consider these two methods to be the best for the determination of recruitment and find that the results of the two tests can be superimposed on each other. They found recruitment to be absent in otosclerosis, syringobulbia, acoustic tumor, and Ménière's syndrome but noted a considerable difference limen in suppurative middle-ear disease.

In a scientific investigation of pitch discrimination Pikler and Harris [17] have presented additional material concerning pitch discrimination in patients with monaural deafness of the recruiting type. Equal loudness does not always yield equal pitch sensitivity. Judgment as to pitch and intensity can be varied with artificial perceptive deafness by administering noise up to 120 db. The information may be of use in selecting hearing aids.

Jerger [18] investigated the influence of stimulus duration on the pure-tone threshold during recovery from auditory fatigue. Twelve young adults with normal hearing received a thermal noise to fatigue the ear. The pure-tone threshold reached the preexposure threshold gradually as time elapsed after the fatiguing stimulus.

Ranke [19] describes the latest findings in the physiology of the cochlea. Mention is made of the vibrating movements of the basilar membrane and of Reissner's membrane, the adaption curve, and cochlear potentials. There is a discussion of the innervation of the sensory cells and the involvement of the fibers and various neurons by auditory stimuli.

Ferrari [20] conducted experiments on the effect of hyperoxygenation with respect to the auditory threshold of various subjects. He reviews the literature related to the subject which deals entirely with hypo-oxygenation; it is inconclusive but does suggest alterations by this technique in certain abnormal states, such as presbycusis, vascular conditions of the ear, and recent head injuries with acoustic lesions. The technique of the author was to have the subjects breathe pure oxygen for 15 to 20 minutes and then to make the audiogram at once; audiograms were also made prior to the experiment. Groups of normal subjects, patients with vascular conditions, patients with presbycusis, and a group which had been subjected to experimental acoustic trauma as well as a group of diabetics with hearing abnormalities failed to give significant alterations. A group of 10 patients with head injuries presenting auditory deficits was also tested. Of these, five whose injury antedated the time of testing by a year or more

gave inconclusive results; the others, however, showed a significant rise of 10-15 db. under the conditions of the experiment. It is suggested by the author that such a test might be used during the evolutionary phase of auditory trauma due to head injury to predict the limit of the final damage to the hearing.

A study of comparative results in cases of speech loss due to otologic injuries as computed by means of various methods from the pure-tone audiograms as well as by means of speech audiometry was undertaken by deStefani.[21] Forty-seven subjects with various grades of hearing loss were tested; they were mainly cases of head injury but included some cases of acoustic trama. Methods tested included the A. M. A. method of 1942, the method of Fowler and Sabine of 1947, the well-known and early method proposed in 1940 by Fletcher of multiplying the decibel loss by 0.8 using certain frequencies, as well as a later method published in the *Acta oto-laryngologica* in 1950; some other formulas were also tried, including one of Bocca and Pellegrini which applies weighted frequencies to Italian speech. The author concluded that the second method of Fletcher and the method of Pellegrini gave satisfactory results in those cases where there were no marked irregularities in the pure-tone curve and which did not present a severe impairment. The other methods presented a difference of 10%-20% between the results obtained by computation from the pure-tone audiogram and those obtained by speech audiometry. A rapid but fairly comprehensive review of previous literature on the subject is included.

Higbee[22] describes a simple method of testing bone conduction in the office which will establish thresholds from the forehead and ensure a static pressure. A headband made of a rubber belt with a small inflatable bag and self-locking buckle is used. This is placed on the head, cinched up loosely, just enough to keep it in place, with the center of the bag opposite the center of the forehead. The vibrator is then placed under the bag, which is connected to a sphygmomanometer and inflated to a pressure of 40 mm. The tubing is then compressed tightly close to the headband and the other connections are removed. The equipment for masking is then added, with earphones placed over both ears. A pad is placed under the earphone of the ear being tested to hold it away from the ear to eliminate the effect of a closed canal on hearing. A pressure of 40 mm. was selected because it is comfortable for the patient, is sufficient to secure the vibrator firmly, and is also low enough to act as a buffer against vibrations being transmitted by the headband. It was found that the difference between the forehead and the mastoid threshold is about 10 db. In the audiogram an adjustment for each frequency may be made by elevating the bone line 10 db. to correspond with the value of thresholds from the mastoid position. Selective masking for each frequency is preferred. This can be done by adjusting the masking noise so that each pure tone is heard at threshold in the ear under test. This bone-conduction test ensures a duplication of position and static pressure which should increase the reliability of the test.

Cochlear potentials in the evaluation of bone conduction were studied in the cat by Goodhill and Holcomb.[23] Their study shows that present bone-conduction audiometry suffers from variable factors introduced by the transducers themselves, their methods of application, and the characteristics of the soft tissue. They suggest that the headbands of bone-conduction receivers used in audiometry should be made adjustable so that the same pressure is applied in all tests regardless of head size. The pressure should not be less than 400 gm. Receivers used in testing should be designed for that specific purpose. Variation of tissue compliance in the individual human subject may cause an error of 5 db. or more in measurements even when pressure is constant at 400 gm. The writers suggest that the compliance can be measured by a simple device which has

been used to measure the compliance of rubber and other elastomers. This device which measures compliance on the "Shore A" or "ducometer" scale may prove feasible to calibrate directly in decibels of correction.

Statten and Wishart [24] discuss two tests which are available for testing the uncooperative child and the preschooler, namely, the psychogalvanic skin resistance (PGSR) test and the peep show. The equipment used and their technique are described in detail. An intensive study was made of 122 PGSR test in 101 children, over an 18-month period. They found that PGSR testing is difficult and may be an unpleasant experience for the child. It requires an experienced team in which every member is aware of the principles involved, knows the limitations of the technique, and has a thorough understanding of the handling of the child. They condemn the indiscriminate distribution of this complex equipment under the assumption that it is a practical method of testing the hearing of any child by an operator. In nearly all cases in which PGSR testing was performed it helped in forming an opinion of the child's hearing. This opinion was reached in most cases by combining the graphic response and the clinical judgment.

Peep-show audiometry has been the most useful method of testing the hearing of the preschool child. It can be performed by one person. The apparatus and technique of the test are described in detail. The Peep show is in the form of an attractive doll's house containing an audiometer and a projector. The test also demonstrates to the observant parent the child's hearing capacity for pure tones. With the type of machine used, the child is attracted, his attention is held, he has no distractions, and he teaches himself to obtain the results the examiner desires. Repeat tests are done even more enthusiastically. This conditioned test, however, is also limited in its use by the age, intelligence, and cooperation of the patient. Every child over 18 months of age is introduced to the peep-show testing. Records of 204 consecutive children tested are reviewed. Two children under the age of 2 of a group of 27 gave satisfactory audiograms. Also, 43% of the 2-year-olds, 80% of the 3-year-olds, 93% of the 4-year-olds, and 90% of those 5 and over gave satisfactory results. Those who could not be conditioned were found to be severely deafened and without appreciation of the sound stimulus. Also, tests on children with low intelligence, brain damage, or severe behavior problems were often classed as failures. Children, if old enough, are routinely tested with the peep show. When the results are unsatisfactory or if further information is desired, the PGSR test is used. The two methods of conditioning in the testing of hearing and their application in obtaining the pure-tone threshold audiogram are thoroughly described. The PGSR does not require the patient's cooperation, but the peep show, which is adapted to the child's ability to respond, must have the patient's cooperation.

Wilkerson and Doyle [25] studied a group of 151 children with impaired hearing in the age range from 6 to 14 years to determine a possible relationship between etiology of hearing loss and the resultant audiometric pattern. Their study gives support to the concept that causes of hearing impairment can be more definitely related to audiogram patterns. The largest etiologic group, which consisted of 68 cases with otitis media and enlarged tonsils and adenoids producing middle-ear impairments, was significantly associated with the moderate loss for all tones and slightly rising audiometric contour audiograms. In the next largest etiological category, consisting of 30 cases of inflammatory neuritis, the cases were distributed among eight audiometric pattern groups. The largest number of audiograms, 16, were characterized by gradual and marked high-tone loss and trough-shaped curves. Each of these patterns represents primarily a perceptive-type loss. Fourteen cases representing loss due to heredity revealed either a moderate loss at all frequencies or marked high-tone loss. Eight out of ten cases in

the birth-injury group revealed a marked high-tone loss. Four of the six cases due to rubella showed a moderate loss for all tones or a trough-shaped curve. The writers believe that a larger sampling of the various etiologic groups would yield results that could be substantiated.

Gundrum [26] made a comparative study of audiometry in simulated and actual partial deafness to ascertain the correctness of the opinion that audiometric tests repeated at various time intervals constitute a reliable basis on which to detect malingering. Repeated audiograms obtained from a group of 50 normal graduate and student nurses instructed to feign partial loss of hearing were compared with those of 50 accident cases showing actual partial deafness. In the first group, with the exception of two cases, the subjects were unable to repeat a tone threshold approaching that of the first audiogram. On the other hand, the tests of the second group revealed relatively small variations between the two audiograms. From these results it appears that repeated audiometric tests are a reliable method for determining actual or simulated partial loss of hearing.

Johnson et al.[27] discuss the problem of functional deafness, which includes the entire group of nonorganic hearing acuity handicaps ranging from classical hysterical and psychogenic deafness to wholly conscious, purposeful, and willful malingering. In a study conducted by the authors on 500 veterans under 60 years of age who were referred for problems relating to service-incurred hearing disability, the incidence of functional deafness varied from 11% in 1951 to 45% in 1954. The increase from 11% to 23% between 1951 and 1952 was due to an increased awareness of and experience with the problem on the part of the clinical team. The marked increase from 23% in 1953 to 54% in 1954 was attributed to the use of a psychogalvanic instrument plus the referral of a large group of veterans for compensation examination during this period. An analysis of this group revealed that functional hearing loss is most frequently an overlay to an actual organic loss rather than to normal hearing. The continuing monthly compensation policy and the use of the classic conversational voice test tend to encourage the development of functional deafness. The diagnosis of this condition and the determination of organic thresholds are most effectively accomplished through the combined efforts of the otologists and audiologists. Signs and indicators of this condition are found as commonly from discrepancies between medical and acoustical examinations as from either examination separately. The threshold of organic hearing acuity can be determined in 95% of the cases of functional deafness by experienced clinical teams with versatile and modern equipment. Significant findings for a diagnosis of functional deafness for all the types of tests employed are given in outline form in this article. Most persons with a functional deafness, whether from malingering or hysteria, respond in essentially the same manner to pure-tone or speech tests. The authors refer to the saucer audiogram described by Doerfler as being diagnostic. An understanding of this finding is very essential in working with nonorganic hearing loss patients. Doerfler noted that this audiometric configuration is usually found between 50 and 90 db. and was present in 80% of the cases diagnosed as functional. He pointed out that persons with normal hearing manifest a comfortable loudness curve for pure tone which corresponds roughly to the 60 db. equal loudness curve. A person simulating a hearing loss will usually take as his reference level something approximating a comfort level. So a person with normal hearing simulating a loss of hearing frequently will have a functional loss which corresponds to the 60 db. equal loudness contour. The writers have found that this occurs in people whose organic thresholds are within the limits of normal or who have an overlay to a conductive loss. However, in those who have an overlay to a high-tone or perceptive organic loss, they

have found that the functional curve falls off rather than rises in the high frequencies. The difference in the shape of these two curves is the result of recruitment. In discussing the value of psychogalvanic skin resistance audiometry, the writers feel that it is an excellent tool and particularly useful with patients having some functional hearing loss. They state, however, that the level of compensation or the rating of the percentage of disability should not be based as a general practice on those results alone unless supported by other test evidence. Proper counseling and adequate education relating to hearing loss and the problems of the hard of hearing given early in the history of the disability would discourage the development of nonorganic hearing loss.

Silverman and Hirsh [28] discuss the problems related to the use of speech in clinical audiometry. They point out some of the shortcomings of speech as it is used now in the various tests for determining auditory acuity. In the measurement of discrimination loss for diagnosis they find that speech is important not as a practical kind of sound, but as a particular kind of auditory stimulus whose properties one is not yet able to specify. For assessment of social adequacy it is the practical feature of speech that is the basic factor in our choice of material for a test which ultimately must be validated in the field. The writers believe that we need to think in terms of different tests for different purposes. Since every-day hearing is characterized by the hearing of speech, test material should be used which is representative of every-day speech. The Armed Forces-National Research Council Committee on Hearing and Bio-Acoustics (CHABA) appointed a working group to formulate a set of criteria for representing every-day speech. The criteria which this group suggested for this speech material are given in detail in this paper. At the Central Institute for the Deaf a set of 100 sentences has been constructed and recorded to conform with the criteria suggested by the working group. This new test may add new information and is definitely necessary to carry out the study in order to have an adequate reply to the question of validity.

Goetzinger and Proud [29] report on the use of speech testing by bone conduction at the University of Kansas Medical Center. They have found this to be a useful adjunct to the regular tests employed. Speech reception tests by bone conduction correlate well with the average pure-tone threshold by bone conduction for frequencies 500, 1000, and 2000 cps. The test has been used as a check upon the reliability of the pure-tone bone-conduction threshold. It has been particularly useful in contributing to diagnosis of conductive impairment, especially when it has not been possible to obtain reliable information by pure tones. It is also an aid in predicting the result of the operative procedures for otosclerosis, where accuracy in determination of the cochlear involvement is paramount.

Pattee and Cary [30] describe a method of converting an existing one-room office space into a satisfactory sound-treated room for speech audiometry, including free-field testing of hearing aids. An inexpensive sound-treated box to enclose the record player is used instead of a second sound-treated room. The results obtained by free-field testing compared very satisfactorily with those obtained by binaural headphones, except in normal hearing persons.

Juers [31] discusses the diagnostic significance of inconsistencies between pure-tone threshold and hearing for speech. Pure-tone speech hearing discrepancies are not present in pure conduction deafness. On the other hand, a greater loss for speech than for pure tones is rather common in nerve deafness. A marked pure-tone speech discrepancy is usually obvious on comparison of the pure-tone audiogram with the patient's ability to understand conversational voice. Calibrated speech tests are necessary to establish a more exact determination of speech hearing capacity. Poorer hearing for speech than for pure tones is found in cochlear hydrops and some instances of prebycusis. A greater loss

for pure tones than for speech, which is less common, is highly suggestive of psychogenic deafness.

Roach and Carhart [32] undertook a study to determine if it is possible to establish a clinically acceptable reference level for bone-conduction audiometry by comparing air- and bone-conduction responses yielded by subjects with sensorineural loss. Standard audiometric tests by air and bone conduction were administered to 23 cases diagnosed as having pure sensorineural loss. The corrections required by the bone-conduction system of the audiometer were computed to the nearest 5 db. These corrections were then confirmed by testing 127 normal listeners in a sound-proof chamber of research caliber. A useful set of reference values for the bone-conduction system was thus obtained with an empirical procedure utilizing the responses of selected cases with hearing loss. The procedure offers the examiner a method for achieving a reasonable calibration of his bone-conduction system.

Van Dishoeck [33] discusses continuous, or sweep-frequency, audiometry. He uses a specially built audiometer known as the Peekel. In this audiometer the differences of sensibility of the normal ear are compensated so that the zero isophone of a normal patient is straight from 200 to 8000 cps. The frequency of 1000 cps is in the middle of the approximately logarithmic scale. One hundred fifty degrees to the left are the frequencies from 80 to 1000 cps and one hundred fifty degrees to the right the frequencies from 1000 to 1200 cps. The frequency is within 3% of the value indicated on the audiogram chart. The instrument is so constructed that frequency calibration is not required prior to use. The decisions of the patient about hearing or not hearing the sweep tone are directly noted on an audiocard fixed to the audiometer. This saves much time. Masking in continuous audiometry is more accurate, as the intensity of the masking noise can be easily adjusted to the level of the flat tone. This apparatus is very suitable for screening tests in school children, factory workers, and soldiers. Early detection and exact registration of an acoustic trauma are only possible with this procedure. In comparison with other techniques continuous audiometry gives more and exact information in less time. The writer suggests it be called "high-fidelity" audiometry.

Corso [34] made a study to determine the effects of two audiometric testing methods on absolute thresholds of hearing. The two testing units used were an ADC audiometer, Model 50-E2, and a Békésy-type audiometer, Reger Model. The threshold data were obtained by the method of limits and the method of adjustment at selected frequencies between 250 and 8000 cps. One hundred five male and female subjects between the ages of 17 and 25 were tested prior to the first audiometric test. All were given a thorough otologic examination. From 250 to 2000 cps, inclusive, the method of limits produces significantly less variability in absolute thresholds among subjects than either the "midpoint" or "peak" audiogram readings in the method of adjustment. For the frequencies tested there is no consistent trend for the mean absolute thresholds obtained by the method of limits to be either significantly larger or significantly smaller than the values obtained by "peak" audiogram readings in the method of adjustment. There is a statistically significant but rather low positive correlation between individual thresholds obtained at a given frequency by the method of limits and those obtained by the method of adjustment, using either "midpoint" or "peak" readings on the Békésy-type audiograms.

The hearing range from threshold to the beginning of nonlinear distortion was measured in both normal and pathological ears by Lawrence and Yantis.[35] There were extensive individual differences in the normal ear for the ranges tested, namely, 1000 and 2000 cps. These are the same as reported earlier. This overload threshold measurement technique was employed in abnormal ears. Middle-ear pathology does not intro-

duce nonlinear distortion into the conductive system. The ossicular system is capable of responding over the same decibel range without overloading. Frequency distortion is present and is reflected in the changed shape of the sensitivity curve. In inner-ear involvement the threshold of overload is lowered and the range from threshold to overload is markedly restricted. There is a relation to the recruitment phenomenon.

Butler and Albrite [36] studied a group of perceptively deafened persons and a group of conductive type hearing loss persons to determine the pitch discrimination function of the pathological ear. Pitch difference limens were measured at 500, 1000, 2000, 3000, and 4000 cps. The performance of the perceptively deafened group was significantly inferior to that of the conductive group at 3000 and 4000 cps. Extensive training served to reduce the magnitude and group variability of the difference limens, but the significant differences between groups remained.

Yantis [37] presents the published information on the results of recruitment in patients with various types of hearing impairment. The accumulated evidence has appeared sufficiently consistent to make the established existence of recruitment a reliable aid in verifying the locus of the lesion causing perceptive losses. The overwhelming evidence in the literature supports the conclusion that loudness recruitment occurs only in lesions involving the sensory, end-organ and is not expected to be present in lesions involving only the eighth cranial nerve and the higher acoustic pathways.

Miller [38] studied a group of 23 cases of cleft palate, ranging in age from 3 to 23, to determine the incidence, type, and significance of hearing losses in these cases. In 12 instances there was a conduction loss affecting the high frequencies only, probably due to lymphoid tissue in the nasopharynx. In severe cases the conduction hearing loss was throughout the entire range tested. It is felt that the high incidence of conduction losses of hearing is due to the presence of the cleft in the hard and soft palate, permitting the entrance of organisms producing upper respiratory infections and their complications. Also, the incomplete closure of the nasopharyngeal port is another means by which organisms can gain access to the body. A large number of the cases have prolific lymphoid tissue in the nasopharynx which occludes the Eustachian tubes, causing a hearing loss. Also, the scarring and distortion of tissues at the nasopharyngeal orifice of the Eustachian tube following surgery may be a cause for the high incidence of hearing loss in these cases. Since this type of hearing loss usually first affects the frequencies above the speech range, the usual tests used in the public school systems may not disclose this. Only a complete individual audiometric test involving all frequencies up to and including 12,000 cps will detect this loss. It is important that all agencies coming in contact with this type of case be aware of this situation so that early recognition of this hearing loss and proper medical treatment can be instituted early to either correct or prevent further progress of the condition.

Holmes and Reed [39] report on the hearing and deafness in a group of 26 cleft-palate patients. Sixty-two per cent of this group had a hearing loss of greater than 10 db. The losses were all of the conductive type and due to chronic middle-ear changes in the form of chronic disease from perforations or from fibrosis and adhesions in the middle ear. No relationship could be found between this hearing loss and the age of closure of the palate, the degree of flexibility of the closed palate, the amount of nasopharyngeal occlusion, or the severity of the initial deformity. Nor did the presence or absence of tonsils or the amount of adenoid tissue bear any relationship to hearing loss. The only practical means of controlling and preventing this hearing loss is that of prompt recognition and early treatment of all ear infections.

Halfond and Ballenger [40] report their audiologic findings in a group of 69 cases

of cleft lip and cleft palate. Seventy-five per cent had some hearing loss, and more than fifty per cent had hearing losses that were significant by medical definition and required special consideration because of possible communication problems. The impairment was of the conductive type. The importance of early and repeated audiometric evaluations in these cases is emphasized.

Fox[41] discusses occupational deafness. The hearing loss may occur suddenly as the result of direct traumatic injury or of explosions, blasts, or gunfire. It may also occur gradually as the result of prolonged exposure to hazardous noise levels. Audiograms made before and during the course of employment are essential to a program of conservation of hearing and for medicolegal purposes. The physician consulted in disputes about cases of allegedly occupational deafness is on safe ground when he confines himself to statements of demonstrated fact and avoids suppositions. The otolaryngologist is especially qualified to collect the facts needed to conserve the hearing of workers and to prevent economic loss to employers from ill-founded claims. He should meet the challenge of occupational deafness by active interest and participation in the various aspects of the problem.

The effects of the tympanic muscle reflexes upon sound transmission were investigated by Weaver and Vernon.[42] Experiments were carried out on the cat. The reflex action was elicited by tonal stimulation of the opposite ear. For excitation a little above reflex threshold there is a reduction in the transmission of low tones. This effect diminishes as the frequency is raised, and disappears around 600 cycles; it then increases between 600 and 1000 cycles. For higher frequencies the tensor tympani produces moderate reduction in transmission, while the stapedius produces only slight effects.

When acoustic stimulation is increased, the muscle action becomes more vigorous and transmission changes progressively greater. For low tones the reduction of transmission keeps pace with the stimulation, so there is a check on the magnitude of stimulation reaching the inner ear. This protects the sensory cochlea against damage until a limit of muscular action is reached. Regulatory effect may be as great as 20 db. for lower tones.

Albrite et al.[43] studied the cochleopalpebral reflex in normal and hard of hearing persons. Four pure tones, 500, 1000, 2000, and 4000 cps, were used as the auditory stimuli. The recording of the reflex and the presentation of the stimuli were placed under automatic control. The intensity was increased by 5 db. for each group of 10 regularly spaced trials until the subject demonstrated an eyeblink response on 5 out of 10 stimulus presentations at a given intensity level. The eyeblink responses for the normal and perceptive groups were quite similar. Differences were found for the intensity of sound required to elicit the reflex. The perceptive group required significantly greater intensity at 500, 1000, and 2000 cps. No differences were found between the groups for the 4000 cps frequency. The patients in the conductive impairment group failed to demonstrate the cochleopalpebral reflex at any of the test frequencies. These results indicated that the eyeblink response test can differentiate between the normal hearing and the perceptively and conductively deafened groups. They suggest that this reflex might be useful as an indirect measure of recruitment.

An attempt is made by Christiansen[44] to present a predictive test with white noise as a fatigue-producing factor. Fatigue was measured by determining the threshold shift at 4000 cps and by measuring recovery time. Arbitrarily chosen were three minutes and 105 db. for the duration and intensity, respectively, of the fatigue-producing factor. The author used the following criteria in his testing:

Threshold shift ½ minute after noise has ceased ≧ 25 db.

Threshold shift 15 minutes after noise has ceased $\geqq$15 db.

Recovery time $>$15 minutes.

What appears to be a simple test for recruitment is suggested by Moulden.[45] After quoting other methods of determining recruitment, he describes his relatively simple one. Forks of 256, 512, and 1024 d. v. are used; the author prefers 512.

The fork is placed on the vertex at maximum intensity for about two seconds, then removed; the patient is asked where the sound is heard. If the sound is heard in both ears, the test is repeated with a smaller intensity of vibration. If the sound is then lateralized, recruitment exists in the nonhearing ear. If recruitment is not demonstrated by one fork, other frequencies should be tried. The test requires a minimum of apparatus and can be carried out in the home.

Jarvis[46] raises the question of where does the phenomenon of "beats" occur. A beat is heard when two pure tones differing slightly in pitch are heard simultaneously. With the use of an audiometer in one ear at, say, 125 d. v. and a fork at 128 d. v. at the other ear, beats are heard. This raises the question as to where the interference takes place. When one ear is deaf the phenomenon does not occur at the low intensity used. The author concludes that the interaction must be central.

Flottorp[47] experimented with spoken and whispered voice in various examining rooms and came to the conclusion that different acoustic conditions in various examining rooms made it impossible to establish any standards for free-field tests based on the spoken or whispered voice. Results of such tests carried out in different places are not comparable.

Varying factors and different examiners make the situation even more questionable.

Acoustic properties in different rooms, different sets of test words, and different examiners were analyzed.

The free-field test may render a good orientation of a patient's hearing ability.

Fournier[48] reports on a continuation of previous work with the difference limen for frequency. This was performed routinely as a part of the audiometric examination by means of a frequency modulating apparatus. His conclusions were as follows: 1. There is a constant association between an increase in the difference limen for frequency and disturbances of pitch perception; in the majority of patients a lowering of pitch perception occurs but sometimes the opposite has been noted. In the same patient, however, the difference limen for frequency and the pitch perception vary in a uniform way. 2. High values for the difference limen, i. e., poor discriminative ability, are associated with more or less impaired intelligibility for speech and would thus affect the degree to which such a patient would benefit from a hearing aid. It is suggested that in case of bilateral loss the aid should be applied to that ear which shows the lowest frequency difference limen. In cases where this test cannot be made, such as children, Fournier believes that the results to be obtained from a hearing aid cannot be predicted in advance regardless of the type of audiometric curve indicated by the usual tests.

Van Dishoeck[49] discusses the screening of school children for hearing. He states that there are many grades between deaf-mute and hard of hearing children and that many of these children are considered dull or mentally deficient unless proper tests are performed. The whispered-voice test he considers useless; audiometric tests in a silent room are essential. Continuous audiometry at fixed intensities with a specially designed audiometer is to be preferred to the usual tests with octave audiometry.

Linhares[50] gives an outline of the methods employed at his clinic in Rio de Janeiro. They are essentially those taught in the departments of otolaryngology and audiology of Northwestern University in this country and include, in addition to the history and complete otorhinologic examination, tuning-fork tests, air and bone

pure-tone audiometry as well as speech audiometry with records. The latter have been adapted to the Portuguese language. Charts and anatomic illustrations from the Northwestern course are presented.

Pietrantoni, Bocca, and Agazzi [51] report on the first results in the conservation of hearing by tympanoplasty. They point out that regardless of whether the operation performed in chronic otitis is a classical or a conservative radical the hearing result is not satisfactory; in a group of 215 cases from various clinics where the latter operation was performed only 52% of the patients attained or kept a hearing level of 30 db. The authors feel that with the new antibiotics and methods of microsurgery on the ear tympanoplasty can achieve simultaneously the healing of centers of osteitis as well as the restoration of hearing. The fundamental principle is the reconstruction of a closed tympanic cavity communicating with the rhinopharyngeal cavity by means of a free Eustachian tube functioning normally; the two windows should recover their full functional capacity. They used the technique and classification of Wullstein, who classifies the operable cases as follows: (1) lesions involving only the tympanum and drum without ossicular damage; (2) lesions of the drum, the tympanum, and the ossicles with the exception of the stapes; (3) total destruction of the ossicles. In the first case, after antroatticotomy through the mastoid with conservation of the tympanic ring, a graft of whole skin is applied so as to obturate the tympanic perforation and cover the epitympanum. In the second case, the bony frame is not respected and the flap of skin is applied directly on the stapes, joining together the edges of the perforation and lining the hollow attic. In the third case, the choice is either to insert a piece of acrylic in place of the stapes or to insert nothing at all. To the usual classification of results, the authors add (1) a table of theoretical results which should be obtained in each case and (2) the social results of the operations. The actual results in 60 cases of all types revealed improvement of hearing in 73%, aggravation in 5%, and unchanged hearing in 17%; according to theoretical considerations (an improvement of 15 db. to be expected for frequencies of 500 to 4000 where the ossicular chain is intact and a postoperative level of not less than 25 db. below normal for the same frequencies where the ossicular chain is interrupted), positive results were 23% and negative ones were 77%. Judged from the social standpoint (a threshold of at least 30 db. postoperatively), 58% of the cases operated on had positive results and 42% negative results. Illustrations of the technique as well as representative audiograms with drawings of the type of preoperative perforations are presented.

Tato [52] discusses the present methods of graphic audiometric notation employed by different authors and proposes an international system of signs for this purpose. The article has to be read in its entirety for comprehension.

Supplement 124 of *Acta oto-laryngologica* consists of an article of 69 pages by Jacques Paul Penn, Ph.D.,[53] concerning voice and speech patterns of the hard of hearing. It is too long for abstracting, but a brief excerpt of the most significant implication derived from this study is the reaffirmation of the existence of a profound relationship between speech and hearing. Neither of these faculties alone would serve a biologically useful purpose (no doubt, he refers to humans alone). Neither could have developed independently in the course of human evolution.

The author believes that in progressive loss of hearing articulation is likely to become progressively worse; therefore, he recommends that speech rehabilitation should be begun at once. (This probably is intended to apply to children, but he does not so state.)

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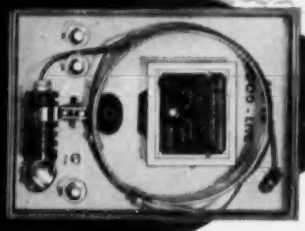

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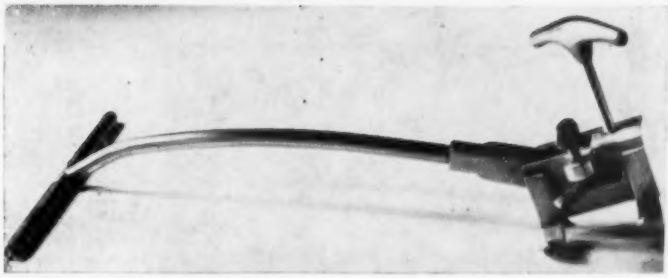

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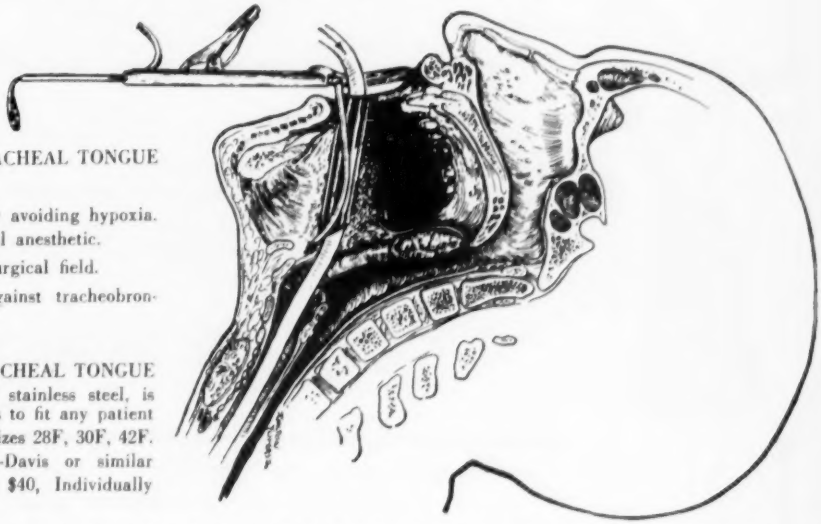

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